TECHNICAL REPORT 008

The Influence of Range Practices on Waterborne Disease Organisms in Surface Water of British Columbia

A Problem Analysis

2003

BRITISH COLUMBIA

Ministry of Forests Forest Science Program

103-06500/

The Influence of Range Practices on Waterborne Disease Organisms in Surface Water of British Columbia

A Problem Analysis

Reg F. Newman, Tracey D. Hooper,
George W. Powell, and Francis M. Njenga

BRITISH COLUMBIA

Ministry of Forests Forest Science Program

NOTE: The Technical Report Series supersedes the Working Paper Series. Working Paper 64, published 2002, was the last of that series.

The use of trade, firm, or corporation names in this publication is for the information and convenience of the reader. Such use does not constitute an official endorsement or approval by the Government of British Columbia of any product or service to the exclusion of any others that may also be suitable. Contents of this report are presented for discussion purposes only. Funding assistance does not imply endorsement of any statements or information contained herein by the Government of British Columbia.

National Library of Canada Cataloging in Publication Data

Main entry under title:

The influence of range practices on waterborne disease organisms in surface water of British Columbia : a problem analysis

(Technical report ; 008)

Includes bibliographical references: p.

ISBN 0-7726-4940-5

1. Range management - Environmental aspects - British Columbia. 2. Waterborne infection - British Columbia. 3. Cattle - Manure - Environmental aspects - British Columbia. 4. Cryptosporidium parvum. 5. Giardia lamblia. I. Newman, Reg F. II. British Columbia. Forest Science Program. III. Title. IV. Series: Technical report (British Columbia. Forest Science Program) ; 008.

SF85.4.C3156 2003 333.74'14'09711 C2003-960069-6

Citation:

Newman, R.F., T.D. Hooper, G.W. Powell, and F.M. Njenga. 2003. The influence of range practices on waterborne disease organisms in surface water of British Columbia: a problem analysis. Res. Br., B.C. Min. For., Victoria, B.C. Tech. Rep. 008. www.for.gov.bc.ca/hfd/pubs/Docs/Tr/Tr008.htm

Prepared by

Reg F. Newman
B.C. Ministry of Forests
Research Branch
515 Columbia Street
Kamloops, BC V2C 2T7

Tracey D. Hooper
1380 Oceanspray Drive
Sidney BC V8L 5J8

George W. Powell
Formerly with
B.C. Ministry of Forests
Research Branch
515 Columbia Street
Kamloops, BC V2C 2T7

Francis M. Njenga
B.C. Ministry of Forests
Chilcotin Forest District
Box 65
Alexis Creek, BC V0L 1A0

Copies of this report may be obtained, depending on supply, from:

Crown Publications
521 Fort Street
Victoria, BC V8W 1E7

Phone: (250) 386-4636
email: crown@pinc.com
www.crownpub.bc.ca

For more information on Forest Science Program publications, visit our web site at www.for.gov.bc.ca/hfd/pubs/index.htm

©2003 Province of British Columbia

When using information from this or any Forest Science Program report, please cite fully and correctly.

ABSTRACT

This problem analysis summarizes the influence of range practices on waterborne disease organisms in surface water within watersheds. The summary is based on literature reviews, case histories, and interviews. Information for the literature review was obtained primarily from scientific journals, and includes reviews and original studies. The disease-causing organisms *Giardia lamblia* (causing giardiasis) and *Cryptosporidium parvum* (causing cryptosporidiosis) are emphasized because of their importance in British Columbia.

DISCLAIMER

Since this document was written, the British Columbia government has announced major changes to the Forest Practices Code and to government ministries, forest regions, and districts. New material on this topic has also been written. Interviews, quotes, and personal communications were obtained from 1997 to 1999 unless otherwise stated. References to government ministries and their geographic entities, references to regulations or guidebooks, and bibliographic citations are current as of 1999.

ACKNOWLEDGEMENTS

We thank Kevin Cameron and Russ Horton for assistance in the production of this publication. We also thank the following for their valuable input: Larry Pommen, Pat Warrington, Rick Nordin, and Ted White formerly of the Ministry of Environment, Lands and Parks, Water Quality Branch; Bill Moorehead of the Interior Health Authority; Corinne Ong of the University of British Columbia Centre for Disease Control; Doug Fraser and Ken Balaski of the Ministry of Forests, Forest Practices Branch; Gillian Arsenault formerly of the East Kootenay Community Health Services Society and Fraser Valley Health Region; Steve Chatwin of the Ministry of Forests, Research Branch; and Barry Boettger of the Ministry of Health Planning, Water Quality Section.

CONTENTS

Abstract ... iii
Disclaimer ... iii
Acknowledgements ... iii
1 **Introduction** ... 1
2 **Objectives** ... 1
3 **Literature Review** ... 1
3.1 Waterborne Disease Organisms of Concern in British Columbia ... 2
3.2 Outbreaks of Waterborne Diseases in British Columbia ... 3
3.2.1 Cryptosporidiosis ... 3
3.2.2 Giardiasis ... 4
3.3 Cattle and *Cryptosporidium parvum* ... 5
3.3.1 Influence of cattle on *Cryptosporidium parvum* oocysts in surface water ... 6
3.3.2 Movement of *Cryptosporidium parvum* oocysts from cattle pastures into surface water ... 8
3.3.3 Influence of cattle on human cryptosporidiosis ... 8
3.4 Cattle and *Giardia lamblia* ... 9
3.4.1 Influence of cattle on *Giardia lamblia* cysts in surface water ... 9
3.4.2 Influence of cattle on human giardiasis ... 10
3.5 Livestock Management and Waterborne Diseases ... 10
3.5.1 Cattle fecal coliform contamination of water ... 10
3.5.2 Factors affecting cattle fecal contamination of surface water ... 11
3.5.3 Protecting drinking water sources from contamination by cattle ... 14
3.6 Other Sources of Waterborne Diseases ... 16
3.6.1 Humans, wildlife, and *Cryptosporidium parvum* ... 16
3.6.2 Humans, wildlife, and *Giardia lamblia* ... 17
4 **Case Histories** ... 18
4.1 Cryptosporidiosis ... 18
4.1.1 Cranbrook 1996 ... 18
4.1.2 Kelowna 1996 ... 22
4.2 *Campylobacter* ... 22
5 **Summary and Information Needs** ... 23
5.1 Livestock Use and the Incidence of Cryptosporidiosis and Giardiasis in Humans ... 23
5.2 Management Actions with Potential to Reduce or Eliminate the Potential Risk of Human Cryptosporidiosis and Giardiasis Linked to Livestock Use ... 24
5.3 Potential Sources of *Cryptosporidium parvum* and *Giardia lamblia* other than Livestock ... 25
6 **Future Research** ... 25
7 **Conclusions** ... 26
References ... 26

APPENDICES

1 Interviews and Interviewees 36
2 Scientific names of wildlife species mentioned in text 44
3 List of personal communications 45

TABLES

1 British Columbian wildlife species thought to be susceptible to *C. parvum* infection 4
2 Outbreaks of waterborne giardiasis in British Columbia between 1980 and 1997 5
3 Correlation of fecal coliform/fecal streptococcus ratios with water contamination sources 11

1 INTRODUCTION

Livestock grazing occurs in many watersheds used for drinking water supplies in British Columbia. The potential for waterborne disease resulting from this activity is a concern because about 86% of British Columbia's population obtain drinking water from surface water sources (B.C. Ministry of Forests and B.C. Ministry of Environment, Lands and Parks 1996). In fact, since 1985, British Columbia has consistently reported a higher annual waterborne disease incidence than the Canadian average (B.C. Ministry of Health 1997). The reasons for the high incidence of waterborne disease in British Columbia are not completely clear, but are likely related to the reliance on surface water supplies combined with minimal water treatment.

It is poorly understood how forest practices such as livestock grazing contribute to the contamination of surface water. Little information exists and a common understanding among stakeholders is lacking. For example, livestock have been implicated in an outbreak of waterborne disease originating from a community water supply in British Columbia, but this association was not accepted by all agencies (B.C. Ministry of Health 1996). Watershed management is more likely to be successful if there is a sound information base that is commonly accepted.

2 OBJECTIVES

This problem analysis was initiated in response to the lack of a common understanding regarding livestock grazing and waterborne diseases. The general objective is to summarize the influence of livestock grazing on waterborne disease organisms within watersheds. The summary is based on literature reviews, case histories, and interviews. This report includes four components:

1) a review of literature on the possible associations between livestock management and waterborne disease organisms;
2) case histories of waterborne disease outbreaks in British Columbia where range activities were implicated;
3) interviews with experts at the University of British Columbia, and in the B.C. Ministry of Agriculture, Fisheries and Food, B.C. Ministry of Health, B.C. Ministry of Forests, and B.C. Ministry of Environment, Lands and Parks; and
4) a determination of the information needs on the waterborne disease issue.

3 LITERATURE REVIEW

Information for the literature review was obtained primarily from scientific journals, and includes reviews and original studies. Government and other types of reports were used when the information was additional to that found in scientific journals. Recent studies (1980–1999) examining links among range and forestry activities and waterborne diseases were of primary interest. The disease-causing organisms *Giardia lamblia* and *Cryptosporidium*

parvum were emphasized because of their importance in British Columbia. An attempt was made to include all studies located in, or directly pertinent to, British Columbia.

This document includes background information on *C. parvum* and *G. lamblia*, but is not intended to be a comprehensive review of these organisms. Reviews of *C. parvum* are provided by Barer and Wright (1990), Meinhardt et al. (1996), Casemore et al. (1997), and others. A British Columbian summary of *C. parvum* is provided by the Centre for Coastal Health (1996). A review of *G. lamblia* is provided by Craun (1984), while Isaac-Renton (1987) provides a British Columbia-based summary of this protozoan. A review was also provided of the use of bacterial indicators such as fecal coliforms to monitor contamination levels, and of the management techniques employed to reduce fecal coliform contamination.

3.1 Waterborne Disease Organisms of Concern in British Columbia

Waterborne diseases are infectious illnesses, epidemiologically associated with the ingestion of water from a water system. Examples of waterborne disease organisms prevalent in British Columbia include the bacterium *Campylobacter jejuni*, and the protozoa *Giardia lamblia* and *Cryptosporidium parvum*. Many of the common waterborne diseases in British Columbia can originate from both human and animal feces.

Most water systems in British Columbia provide only simple disinfection (mostly chlorination) of water supplies (Willoughby 1993). The risk of a bacterial disease outbreak originating from a water supply is greatly minimized by following recommended disinfection procedures (B.C. Ministry of Health 1982). The protozoa *G. lamblia* and *C. parvum*, however, are not as easily controlled using disinfection alone. Under certain conditions, the control of the infective stage (cyst) of *G. lamblia* requires greater disinfectant contact time and disinfectant residual than that required for control of most bacteria (Jarroll et al. 1984; Jakubowski 1990). The *C. parvum* egg stage (oocyst) is even more resistant to disinfection with chlorine than is *G. lamblia* (Barer and Wright 1990; Rose 1990; Meinhardt et al. 1996). Therefore, it is generally recommended that both filtration and disinfection are required for adequate protection from *G. lamblia* and *C. parvum* (Madore et al. 1987; Barer and Wright 1990; Jakubowski 1990; Rose 1990; Fogel et al. 1993; LeChevallier and Norton 1995).

Water filtration is not widely used in British Columbia, despite the fact that approximately 50% of the water systems in the province draw water from a surface water source (E. Bonham, pers. comm.)[1]. In 1997 there were only 12 water systems in British Columbia that treated surface water beyond simple disinfection, representing about 1% of the systems using surface water. Ten more communities were in the design, planning, or investigation phases for advanced treatment of surface water (B.C. Ministry of Municipal Affairs and Housing 1997).

This report focuses on the protozoa *C. parvum* and *G. lamblia* because these organisms have already caused large disease outbreaks in British Columbia, and have the greatest potential to cause future outbreaks because of their relative resistance to chlorination. Furthermore, *G. lamblia* was identified as the most frequent agent in waterborne outbreaks in the United States (Rose 1988; American Water Works Association 1994), and is reported as the most common human gastrointestinal parasite in British Columbia

1 For complete information on personal communications, see Appendix 3.

(Isaac-Renton 1987). *C. parvum* is also a concern because there is currently no effective treatment for the disease caused by this organism (Smith and Rose 1990). Bacterial diseases, such as those caused by *Campylobacter* and *Escherichia coli* O157:H7, were not emphasized in this review because outbreaks of bacterial waterborne disease can likely be minimized if disinfection systems are properly used. Nonetheless, bacterial contamination of surface water used for drinking water supplies deserves some consideration because about 10% of the water systems in British Columbia do not chlorinate (E. Bonham, pers. comm.). It is also well known that an increased concentration of bacteria and suspended sediments in untreated water poses a greater challenge to the water treatment process, thereby increasing the chances of water treatment failure. The outbreak of *E. coli* O157:H7 infection associated with a municipal water supply in Walkerton, Ontario in 2000 was reported to be caused by a water treatment system that was overwhelmed by increased bacteria and turbidity (Bruce-Grey-Owen Sound Health Unit 2000).

3.2 Outbreaks of Waterborne Diseases in British Columbia

Between 1980 and 1997, there were 22 recognized outbreaks of waterborne diseases in British Columbia. Four more outbreaks were suspected or were under investigation during that period. The number of laboratory-confirmed cases from all outbreaks ranged from fewer than 10 to 157, but the number of suspected cases ranged from 2097 to 10 000 (B.C. Ministry of Health 1997). Thirteen of the outbreaks were attributed to the protozoan *G. lamblia*, five to the bacterium *Campylobacter*, two each to the protozoan *C. parvum* and the bacterium *Salmonella*, and one to the protozoan *Toxoplasma*. The causes of four outbreaks were not identified. Cattle were implicated in the two outbreaks attributed to *C. parvum*, and in one outbreak of *Campylobacter*. There were no associations made between range or forestry activities and any of the outbreaks due to *G. lamblia* (B.C. Ministry of Health 1997).

An April 1998 waterborne outbreak of cryptosporidiosis in Chilliwack, B.C. was attributed to contamination of the surface water supply by wild animals as well as by visiting domestic animals (dogs and horses). Cattle and sheep on a nearby ranch tested negative for *C. parvum* (Fraser Valley Health Region 1998).

3.2.1 Cryptosporidiosis Cryptosporidiosis is the disease caused by *C. parvum*. Cryptosporidiosis typically causes gastrointestinal illness in humans and animals, and is a leading cause of diarrhea in humans and cattle (Mann et al. 1986; Smith and Rose 1990; Meinhardt et al. 1996). In immunocompromised humans (those with impaired immune systems), infection of the biliary and respiratory tracts can also occur (Soave and Armstrong 1986). In such individuals, the disease can be fatal (Barer and Wright 1990).

Wallis et al. (1996) reported about 2.5% prevalence (n=720) of *C. parvum* oocysts in untreated and treated drinking water samples collected throughout British Columbia. The prevalence of cryptosporidiosis in British Columbia is difficult to determine, however, because gastrointestinal illnesses are often under-reported, and cryptosporidiosis became a reportable disease for humans only in 1995 (Centre for Coastal Health 1996). In that year, 196 cases were reported. The highest number of cases occurred in the Kootenays (20–29 cases/100 000 population) and the North Okanagan (15 cases/100 000 population)(Centre for Coastal Health 1996). In North America, incidences of the disease have been reported from areas supplied with water from watersheds protected from human activities as well as from communities supplied by unprotected watersheds (Rose et al. 1997).

Three large outbreaks of cryptosporidiosis have been recorded in British Columbia, two of which were associated with contaminated drinking water (B.C. Ministry of Health 1997). In 1990/91, an estimated 89 people in the Lower Mainland contracted cryptosporidiosis following an outbreak in a community swimming pool (Bell et al. 1993). In 1996, an estimated 2097 people in Cranbrook and 10 000 people in Kelowna contracted cryptosporidiosis through contaminated drinking water (Centre for Coastal Health 1996; B.C. Ministry of Health 1997). The actual number of people exposed to the pathogen could be much higher than those cases reported, however, since only 10.5% of the people exposed to contaminated drinking water in British Columbia are believed to contract cryptosporidiosis (Centre for Coastal Health 1996).

Cryptosporidial infections have also been identified in wolves, deer, marten, deer mice, and a variety of birds on southern Vancouver Island (Centre for Coastal Health 1996). In addition, a number of other British Columbian wildlife species are thought to be susceptible to *C. parvum* infection based on reports from outside the province (Table 1).

TABLE 1 *British Columbian wildlife species thought to be susceptible to* C. parvum *infection (Source: Centre for Coastal Health 1996)*

Common name	Scientific Name
beaver	*Castor canadensis*
black bear	*Ursus americanus*
black rat	*Rattus rattus rattus*
cottontail rabbit	*Sylvilgagus nuttalli*
coyote	*Canis latrans*
deer mouse	*Peromyscus maniculatus*
house mouse	*Mus musculus domesticus*
marten	*Martes americanus*
mule deer	*Odocoileus hemionus*
Norway rat	*Rattus rattus norvegicus*
raccoon	*Procyon lotor*
red fox	*Vulpes fulva*
striped skunk	*Mephitis mephitis*
white-tailed deer	*Odocoileus virginianus*
wolf	*Canis lupus*

3.2.2 Giardiasis *G. lamblia* are pathogenic organisms that can cause a gastrointestinal infection known as giardiasis in humans and in some animals. In humans, symptoms of giardiasis typically include abdominal pain, bloating, diarrhea, and malabsorption (faulty absorption of nutrients from the alimentary canal). Infection can occur after ingestion of as few as 10–25 infective cysts (Rendtorff 1954), and can last up to 3–4 months (Isaac-Renton 1987). Immunity to the disease can be acquired, and can last at least 5 years (Isaac-Renton et al. 1996).

G. lamblia is the most commonly reported human gastrointestinal parasite in British Columbia (Isaac-Renton 1987). Although more than 1000 cases of giardiasis are reported annually in British Columbia, more cases undoubtedly go unreported because not all infected individuals show signs of the dis-

ease (Isaac-Renton et al. 1996). Between 1980 and 1997, there were 13 known outbreaks of waterborne giardiasis in British Columbia (B.C. Ministry of Health 1997). Twelve of these outbreaks occurred in the southern part of the province, mainly in the Kootenay and the Okanagan regions. Dates and locations of outbreaks in British Columbia are given in Table 2.

Wallis et al. (1996) reported about 17% prevalence (n=720) of *G. lamblia* in untreated and treated drinking water samples collected throughout British Columbia. *G. lamblia* appears to be common in community drinking water supplies in the province. In a 12-month province-wide survey, Isaac-Renton et al. (1996) examined 244 water samples (153 untreated, 91 treated) from 86 sites. *G. lamblia* was found in 64% of all water samples (68% untreated, 59% chlorinated), and at 69% of all sample sites. There were many potential sources of contamination around the sample sites. For example, none of the sites had restricted public access, many were near agricultural areas, and eight were downstream of small villages or individual residences.

G. lamblia has also been found in wild animals in British Columbia. In one study, almost 14% (n=388) of animal fecal samples tested contained *G. lamblia* cysts. The highest levels were found in muskrats (40%; n=20), followed by beaver (14.7%; n=299), and deer (7.1%; n=14). No cysts were found in samples from mink, marten, lynx, bear, or mice (Isaac-Renton 1987).

TABLE 2 *Outbreaks of waterborne giardiasis in British Columbia between 1980 and 1997 (Source: B.C. Ministry of Health 1997)*

Year of Outbreak	Location
1981	100 Mile House
1982	Kimberley
1985	Creston
1986	Penticton (two separate outbreaks)
1987	Kelowna (Black Mountain)
1990	Kitimat
1990	Creston
1990	Fernie
1991	West Trail / Rossland
1991	Barriere
1995	Revelstoke
1996	Valemount

3.3 Cattle and *Cryptosporidium parvum*

Most recorded cases of *C. parvum* in domestic animals have been from cattle (Casemore et al. 1997). The parasite has been recorded in cattle throughout the world (Casemore et al.1997), and is widely distributed throughout North American cattle operations (Mann et al. 1986; Casemore et al. 1997).

Calves that are younger than 3 weeks old are most susceptible to *C. parvum* infections, although the infection can continue up to 3 months of age (Casemore et al. 1997). Infected calves can act as large reservoirs for *C. parvum* (Meinhardt et al. 1996). A single calf can shed up to 10 billion oocysts/day for about 2 weeks (Current and Garcia 1991). Crockett and Haas (1997) claim that one infected calf can produce more *C. parvum* oocysts per day than 1000 infected immunocompromised people. Oocysts are immediately infective upon shedding, and thus can lead to widespread transmission of

the disease, especially where large numbers of cows and calves are confined to restricted areas (e.g., calving or wintering pastures). Adult cows are generally resistant to the disease, but they may occasionally shed small numbers of oocysts (Meinhardt et al. 1996; Casemore et al. 1997).

A study by the United States Center for Disease Control (USCDC) revealed that 40% of beef cattle farms had *C. parvum* on their premises (cited in Centre for Coastal Health 1996). In Manitoba, 26% of the beef herds with diarrhea were infected with *Cryptosporidium* spp. (Mann et al. 1986). That study, however, did not indicate which species of *Cryptosporidium* had caused the infection. Statistics on infection rates of *C. parvum* in British Columbia's beef cattle industry were not found, but a report by the Centre for Coastal Health (1996) suggests that infection rates may be similar to those of the USCDC findings (i.e., 40% of beef cattle farms).

3.3.1 Influence of cattle on *Cryptosporidium parvum* oocysts in surface water A number of studies have implicated cattle with increased *C. parvum* oocysts in surface water. For example, Hansen and Ongerth (1991) compared *C. parvum* oocyst concentrations in water samples collected from two physically similar watersheds in the central Cascade Mountains of western Washington State. In one watershed, commercial timber cutting, watershed management, and sanitation were carefully controlled, and no unsupervised human activity was permitted. In the other watershed, private and public forests were used extensively for logging and recreation, and watercourses received runoff from towns, rural homes, and dairy farms. In this study, the highest oocyst concentration (18.2 oocysts/L) occurred at a site downstream of dairy farms in the uncontrolled watershed. The lowest oocyst concentration (0.2 oocysts/L) occurred in the controlled watershed. The authors believed that the elevated oocyst counts were strongly related to the local dairy farm activities.

Similarly, Rose et al. (1989) found that in a watershed in the western United States, the highest levels of *C. parvum* oocysts (1.09 oocysts/L) were found in a section of river that ran through an agricultural area with cattle, sheep, and chicken farms. Oocyst concentration at this site was double that of a site 19 km upstream at a lake outlet (0.58 oocysts/L). The authors state that the only potential major input of *C. parvum* along the 19-km stretch appeared to be from animal wastes.

In another study, Madore et al. (1987) analyzed surface water samples collected in Arizona. The highest levels of *C. parvum* oocysts (5800 oocysts/L) were found in water from an irrigation canal that ran through a high-density agricultural area with cattle pastures. Slightly lower levels were found in an irrigation canal exposed to treated sewage discharge (5300 oocysts/L). The lowest oocyst concentration was found in stream water exposed to ranchland runoff (0.8 oocysts/L). The authors state that oocysts may be entering the water in fecal material via sewage effluent discharges, septic tank leakage, recreational bathing, agricultural runoffs, or the erosion of soils exposed to infected feces.

Ong et al. (1996) examined the correlation between cattle presence and levels of *C. parvum* in a stream in southern interior British Columbia. Water samples were collected upstream and downstream of a 200-head cattle ranch over a 10-month period. Samples downstream of the ranch had a significantly higher ($P<0.05$) oocyst level (0.133 oocysts/L) than upstream samples (0.056 oocysts/L). The authors do not specifically attribute the increased

oocyst concentration downstream of the ranch to cattle; however, they note that the peak concentrations of oocysts occurred during the height of calving activity in February and March. Fecal samples from cattle on the ranch were not tested for *C. parvum* oocysts during the study.

In contrast to these studies, LeChevallier et al. (1991) sampled 66 surface water treatment plants in Alberta and 14 U.S. states. The authors present data showing no significant difference in *C. parvum* oocyst levels between surface waters from protected watersheds and those exposed to agricultural runoff. In the same study, oocyst levels associated with sites receiving industrial (urban) sources of pollution were approximately 10 times higher than from protected sites. The authors do not state the source(s) of agricultural runoff in this study.

The group of environmental studies reviewed in this section provides variable and sometimes conflicting evidence of the relationship between cattle and *C. parvum* oocysts in surface water. No doubt this is due to the wide variability of stream types, watershed conditions, and human activities in the various studies. The lack of a standardized laboratory methodology (e.g., Atwill 1996; Centre for Coastal Health 1996) and the lack of standardized environmental sampling techniques (e.g., LeChevallier et al. 1991; Atwill 1996) may also add to the variability. Nonetheless, there seems to be a sufficient correlation between increased levels of oocysts in water and the presence of cattle to justify further examination of cattle as a source of oocysts in surface water.

Using the limited evidence presented, it appears that the type of cattle management may influence the degree of oocyst contamination. For example, Madore et al. (1987) found the highest concentration of oocysts (5800 oocysts/L) in surface water that ran through agricultural areas with cattle pastures, while Hansen and Ongerth (1991) found the highest oocyst concentration (18.2 oocysts/L) at a site downstream of dairy farms. In comparison, Madore et al. (1987) found the lowest oocyst concentration in stream water exposed to "ranchland" (rangeland) runoff (0.8 oocysts/L). On dairy farms and intensively managed beef-cattle pastures, cattle movement is generally confined, resulting in local concentrations of animals. Dairy farms and intensively managed beef-cattle pastures also often rely on forage grown on land seeded to productive, domesticated forage plants. This allows for a greater carrying capacity (an ability to sustain greater densities of cattle for longer periods) when compared to operations in which cattle forage on native plants. A greater carrying capacity of cattle equates to greater manure deposition and potentially greater oocyst concentration in associated surface water. On rangeland operations, cattle are allowed to roam and forage over areas of native plants. The lack of close confinement and the lower carrying capacity of rangeland operations may explain the lower oocyst concentrations found by Madore et al. (1987) in stream water associated with these areas. Although Ong et al. (1996) noted increases in oocyst concentration downstream of a ranch, it is important to note that the area in the Ong et al. study was used for overwintering and calving activities, and not for free-range use.

In a review, Atwill (1996) states that there is incomplete scientific evidence supporting the claim that cattle are a significant source of *C. parvum* in surface water. This author points out that we must first identify the primary quantitative sources of this parasite in the environment before it can be claimed that cattle production is a leading environmental source of infective *C. parvum* for water. Similarly, the group of environmental studies reviewed

in this section does not provide sufficient evidence to confidently state the situations in which cattle are important causes of elevated oocyst concentrations in surface water. In general, the studies do not contain useful quantitative information for watershed management because few details of cattle management were provided. Only the paper by Ong et al. (1996) provides such necessary details as cattle numbers, season of use, and age class of the animals.

3.3.2 Movement of *Cryptosporidium parvum* oocysts from cattle pastures into surface water Mawdsley et al. (1996) provided the only known study examining a potential mechanism for the movement of *C. parvum* oocysts from cattle pastures into surface water. The authors used simulated rainfall applied to intact cores of three soil types. Soil cores were inoculated with 100 million oocysts/core. Artificial rainfall was applied to soils at maximum water-holding capacity, at 70 mL/hour for 4 hours on alternating days for 21 days. This rate is equivalent to approximately 1.6 cm of rain per day for a 16-cm total over 21 days. Therefore, the amount and timing of rainfall is not representative of semi-arid or drier environments, such as in parts of the interior of British Columbia. The authors found that, under the conditions examined, oocysts could leach 30 cm down in two of the three soil types. Surprisingly, vertical transport of oocysts in the clay loam soil was greater than in the silty loam soil, while the loamy sand soil did not allow any vertical transport of oocysts. Sandy soils are generally known to be more permeable than clay or silt soils; however, the authors suggest that macropores derived from natural soil structure may have been an important pathway for the water-mediated movement of oocysts through clay loam soils. When the cores were examined destructively, it was found that the distribution of oocysts was similar in all three soil types. Most oocysts were located in the top few centimetres of soil. This study suggests that oocysts may move through the soil and into the groundwater, as well as by the presumed mechanism of overland transport.

3.3.3 Influence of cattle on human cryptosporidiosis Most of the large outbreaks of waterborne cryptosporidiosis in North America and the United Kingdom since 1984 have resulted from failures in water filtration and treatment procedures, or from sewage contamination of drinking water supplies or public swimming pools (Barer and Wright 1990; Rose et al. 1997). The degree to which humans contract cryptosporidiosis through waterborne sources as opposed to direct human-to-human transmission is unknown (Atwill 1996). Contaminated drinking water may seem to be the primary source of cryptosporidiosis because large outbreaks can occur, which draws much media and public attention. These types of outbreaks, however, occur infrequently (B. Moorehead, pers. comm.). Most cases of cryptosporidiosis, in fact, probably result from person-to-person contact (Rose 1988).

Ong et al. (2002) determined the genotypes of *C. parvum* found in human fecal specimens of cryptosporidiosis cases in British Columbia. The authors reported that *C. parvum* genotypes from the 1996 Cranbrook and 1998 Chilliwack cryptosporidiosis outbreaks matched Genotype 2, previously isolated from human, cattle, sheep, goat, and deer hosts. This gentoype was also found in 19% of the isolates from 150 sporadic cryptosporidiosis cases in the Greater Vancouver and Fraser Valley Regional Districts of B.C. Genotype 2 has been associated with cattle-to-human transmission Ong et al. (2002); however, the study does not provide conclusive evidence that cattle were the

source of the cryptosporidiosis outbreaks studied, since human, sheep, goat, and deer sources cannot be ruled out. The study has demonstrated that *C. parvum* Genotype 2 is an important contributor to cryptosporidiosis in British Columbia and that cattle are a possible source.

Attempts to link cattle with outbreaks of human cryptosporidiosis are complicated by a general lack of knowledge about *C. parvum* ecology. It is significant that Dupont et al. (1995) found that human subjects could contract cryptosporidiosis following ingestion of oocysts produced by calves. However, the rate and degree to which infection occurs following consumption of contaminated drinking water outside of an experimental setting apparently has not been determined (Meinhardt et al. 1996). Rose (1988) also notes that the "inability to determine the potential infectivity of oocysts is one of the limitations of studies on the environmental occurrence of protozoa." Similarly, Meinhardt et al. (1996) state that there currently are no appropriate means of determining the potential pathogenicity or virulence of *C. parvum* oocysts for humans. These authors also note that the inability to determine the medical significance of different isolates in environmental samples is a serious constraint. Simply finding oocysts in water samples does not necessarily indicate an immediate health risk to humans. Rose (1988) notes that the current lack of information on oocyst survivability makes it difficult to determine whether oocysts are, in fact, viable upon entering water treatment facilities. These kinds of limitations complicate efforts to link contamination sources with waterborne outbreaks of cryptosporidiosis.

3.4 Cattle and *Giardia lamblia*

Giardiasis has been identified in cattle in Europe and North America (Buret et al. 1990). In southern Alberta, Buret et al. (1990) found that 10.4% (n=49) of cattle tested were infected with *G. lamblia*. No adults (n=26) were infected, but 22.7% (n=23) of calves tested had cysts in their feces. These rates were probably underestimated, however, because cysts are shed intermittently, and only one sample was collected from each animal (Buret et al. 1990). Similar rates of infection in cattle (10%) have been found in Colorado (Davies and Hibler 1979).

3.4.1 Influence of cattle on *Giardia lamblia* cysts in surface water Rose et al. (1989) found that, in a watershed in the western United States, the highest levels of G. *lamblia* cysts (0.22 cysts/L) occurred in a section of river that ran through an agricultural area with cattle, sheep, and chicken farms. Cyst concentration at this site was more than double that of a site 19 km upstream at a lake outlet (0.08 cysts/L). The authors state that the only potential major input of *G. lamblia* cysts along the 19-km stretch appears to be from animal wastes.

LeChevallier et al. (1991) sampled 66 surface water treatment plants in Alberta and 14 U.S. states. The authors present data showing an increase in *G. lamblia* cysts in areas exposed to agricultural runoff compared to that of protected watersheds. However, the authors do not state the source(s) of agricultural runoff in this study.

Ong et al. (1996) examined the correlation between cattle presence and levels of *G. lamblia* in a stream in southern interior British Columbia. Water samples were collected upstream and downstream of a 200-head cattle ranch over a 10-month period. Samples downstream of the ranch had a significantly higher ($P<0.05$) cyst level (0.090 cysts/L) than upstream samples (0.052 cysts/L). The authors state that their results are consistent with the

view that infected livestock may contribute to parasite contamination. They also note that the peak concentrations of cysts occurred during the height of calving activity in February and March. Fecal samples from cattle on the ranch were not tested for *G. lamblia* cysts during the study.

The studies reviewed in this section are a subset of the studies reviewed for *C. parvum* in Section 3.3.1. The conclusions reached in Section 3.3.1 are generally also pertinent to *G. lamblia*. There is correlative evidence of a link between cattle and increased levels of *G. lamblia* cysts in water, but the evidence is not definitive enough to confidently state the situations in which cattle are important causes of elevated levels of cysts in surface water.

3.4.2 Influence of cattle on human giardiasis Ong et al. (1996) provide evidence of a link between cattle and *G. lamblia* contamination of surface water. It should be noted, however, that although it is suspected that cattle are sources for human giardiasis (Buret et al. 1990), this has not been confirmed (Ong et al. 1996). In fact, cattle have not been implicated in any of the waterborne outbreaks of giardiasis reported in British Columbia since recording started in 1980 (B.C. Ministry of Health 1997). In 95 outbreaks of waterborne giardiasis in the United States, 71% resulted from sewage contamination of surface waters, and another 12% resulted from contaminated groundwater (Wallis 1994). Beaver and other causes (unspecified) were suspected in the remaining 17%. Craun (1984) summarized outbreaks of waterborne giardiasis in the United States between 1965 and 1979. Cattle were not suspected as a cause in any of these outbreaks. In the same summary, grazing sheep were listed as one of several possible causes in a single outbreak in Colorado: "Several active beaver ponds, grazing sheep, and a shepherd were noted in the area."

Finding *G. lamblia* cysts in drinking water supplies in areas of livestock operations does not necessarily indicate that cattle are sources of waterborne outbreaks of giardiasis. Many other warm-blooded species are also known to be vectors. Cattle-derived cysts in such water supplies may not be viable or virulent, or even of the correct species to cause infections in humans. Unfortunately, the ability to determine the role that cattle may play in waterborne outbreaks of giardiasis is currently hampered by monitoring techniques that cannot adequately determine cyst viability, or distinguish between different species of *Giardia* (Jakubowski 1990).

3.5 Livestock Management and Waterborne Diseases

3.5.1 Cattle fecal coliform contamination of water Part of the difficulty in accurately assessing the contribution that cattle can make to *C. parvum* and *G. lamblia* contamination of surface waters is that little is known about the general ecology of these pathogens. There have been numerous studies, however, on associations between cattle management practices and fecal coliform (FC) contamination of water. Many waterborne pathogens are often difficult to trap and culture; consequently, other measures (such as FC concentrations) are used to monitor water quality. Fecal coliforms are easy to detect, simple to culture, and usually associated with other mammalian intestinal pathogens (Gary et al. 1983; Larsen et al. 1994).

There are difficulties, though, in using FCs to monitor water quality when pathogens such as *C. parvum* and *G. lamblia* are the source of contamination. Fecal coliforms are useful indicators of these pathogens only if they behave in the same manner. For example, *C. parvum*, *G. lamblia*, and FCs may all move

differently in the water column, they may have different survival rates, and/or they may have different settling patterns in bottom sediments. Although LeChevallier et al. (1991) found that *G. lamblia* densities were significantly positively correlated with FC levels, FCs are generally considered to be poor indicators for protozoan pathogens such as *C. parvum* and *G. lamblia* (Williams 1981; Rose et al. 1989; American Water Works Association 1994; LeChevallier et al. 1997).

Ratios of FC to fecal streptococcus (FC/FS) concentrations have been used in many studies to identify the primary source of water contamination. Much disagreement exists among researchers about the correlation of these ratios with contamination sources. For example, some studies have suggested that FC/FS ratios less than 0.10 indicate that wildlife are the source of water contamination, while other studies suggest that ratios of 0.08–1.20 implicate cattle as the contamination source (Table 3). The B.C. Ministry of Health does not consider the use of FC/FS ratios to be an acceptable means of identifying water contamination sources.

In this review, studies of cattle fecal contamination of water are presented as a means of illustrating how certain livestock activities and management practices could influence *C. parvum* and *G. lamblia* concentrations in community drinking water supplies. It is acknowledged that many researchers believe that the link between FCs and these pathogens is weak, and that the information presented here may not accurately describe the patterns of these pathogens in community water sources. It is also acknowledged that the applicability of these studies to situations in British Columbia may possibly be tenuous, because all of these studies were done in watersheds in the United States. Until more local studies are done, and until studies on the general ecology of pathogens such as *C. parvum* and *G. lamblia* are produced, the following information may provide the best means of understanding how cattle could contribute to outbreaks of waterborne disease.

TABLE 3 *Correlation of fecal coliform / fecal streptococcus (FC/FS) ratios with water contamination sources*

FC/FS ratio				
Wildlife	**Cattle**	**Cattle and wildlife**	**Human**	**Reference**
<0.05	>0.10			Doran and Linn 1979
<0.10	0.10–0.60		>4.00	Geldreich 1976
		<1.00	>2.50	Gary et al. 1983
<0.04	0.08–1.20	0.04–0.08	>4.00	Tiedemann et al. 1988

3.5.2 Factors affecting cattle fecal contamination of surface water

Cattle behaviour and manure deposition Stocking density, length and timing of grazing period, average manure loading rate, distribution pattern of manure, and time period in which manure is present all affect the potential for cattle to be sources of water pollution (Sweeten and Redell 1978). The effects of manure deposition by free-ranging cattle on water quality were studied by Larsen (1989) in a watershed in central Oregon. Larsen examined:

1) distribution patterns of manure piles throughout areas grazed by cattle; 2) rates of fecal deposition into a stream; and 3) the relationship between distance of manure deposition from the stream and its potential for contributing to water contamination.

The highest concentration of manure piles occurred in meadows used for supplemental feeding during winter. The next highest concentration of manure occurred in the riparian zone. This area provided the only water source, was easily accessible, and had high quantity and quality of forage. The lowest manure concentrations were found far from the stream, and in areas with steep slopes. This indicated that cattle tended to linger in areas with easy access to water. Other studies have shown that cattle drink at least once a day, and travel an average of 5–8 km/day; consequently, cattle activity tends to be concentrated around water sources (Cully 1938; Sneva 1969).

In Larsen's (1989) study, the highest levels of cattle activity in the stream occurred in the summer, while the lowest levels occurred in the fall. The average time that cattle spent in a stream ranged from 11.2 minutes/animal/day in the summer to 2.6 minutes/animal/day in the fall. Correspondingly, the highest number of fecal deposits in the stream occurred in the summer.

Deposition rates were similar among other seasons. The average defecation rate in the stream ranged from 0.17 defecations/animal/day in the winter and spring to 0.41 defecations/animal/day in the summer. In an experiment testing the effects of simulated rainfall on overland bacterial transport, Larsen (1989) found that manure deposited at 0.7 m from a simulated stream resulted in significantly lower FC levels (2.25/L) compared to manure deposited directly in the stream (42.80/L). Therefore, manure deposited away from watercourses likely does not have as great an impact on water quality as manure deposited directly in the water.

Kress and Gifford (1984) also used simulated rainfall trials to study patterns of fecal coliform release from cattle feces. They found that the highest FC releases were from fresh (e.g., 2-day-old) fecal deposits, but FC releases from fecal deposits 100 days old still exceeded recreational water quality standards. Similarly, Thelin and Gifford (1983) found in a simulated rainfall experiment that fecal deposits less than 5 days of age released millions of FCs/100 mL into water, and that fecal deposits 30 days old still released 40 000 FCs/100 mL. The authors concluded that FC concentration in a stream is partially dependent on the length of time since grazing occurred in the area.

Watershed characteristics Watershed hydrology, soil properties, and stream characteristics (e.g., temperature, pH, turbidity, flow rate, sedimentation rate, stream gradient) can all affect FC concentrations in surface waters (Zurbrigg 1992). Tiedemann et al. (1987) also found that watershed topography and vegetation played a major role in FC contamination in watersheds that had been grazed by cattle. In that study, the authors examined FC levels in stream water in response to four cattle-grazing management strategies in 13 forested watersheds in Oregon. They found that water samples from high-elevation sites, sites with steep slopes, or sites with well-forested side-slopes that restricted cattle access to streams generally had lower FC counts than water samples from meadows with open forests that did not restrict cattle movements, or from meadows with riparian zones that attracted cattle. Tiedemann et al. (1987) concluded that FC concentrations in stream water

appeared to be more closely related to watershed characteristics that influence where livestock congregate than to cattle stocking densities.

Runoff and infiltration Water infiltration rates and runoff levels in watersheds are affected by factors such as:

- type, intensity, duration, and distribution of precipitation;
- previous moisture conditions;
- watershed drainage patterns;
- soil physical and chemical properties; and
- vegetation characteristics.

Cattle grazing affects runoff levels in watersheds primarily by causing changes in soil physical properties and in vegetation cover. Removal of vegetation cover and subsequent litter loads through grazing, and soil compaction due to trampling, can result in lower infiltration rates and thus increased runoff (Alderfer and Robinson 1947). High runoff levels are often observed near cattle feedlots, which typically have compacted soils and no vegetation cover (Baxter-Potter and Gilliland 1988).

Doran and Linn (1979) found that FC levels in runoff from a grazed pasture in eastern Nebraska was 5–10 times higher than in runoff from an ungrazed pasture. Similarly, Kunkle (1970) found that FC levels were higher in runoff from a cattle-grazed plot than from a control plot in a Vermont watershed, and that FC concentrations in runoff increased during storms. In the Pacific Northwest, Jawson et al. (1982) studied the effects of cattle grazing on indicator bacteria in runoff by comparing a summer-grazed watershed with an ungrazed watershed. They found that FC levels in runoff were related to the length of time since cattle grazing, but that FCS also persisted in runoff long after cattle were removed from the watershed. Tiedemann et al. (1987) also found that FC levels in an Oregon watershed remained elevated after cattle were removed. These authors related their findings to moisture conditions and drainage patterns in the watershed. They believed that FC levels remained elevated after cattle were removed from the watershed because high stream and overland flows during spring runoff washed fecal material from stream banks into the stream channel.

The role that soil conditions play in transport of cattle fecal material in watershed runoff was examined by Larsen et al. (1994). They used a simulated rainfall experiment to study how infiltration rates of soils surrounding a simulated stream affected bacterial loads in the stream. In trials with sandy (permeable) soils, about 2.2 million FCS from cattle feces were delivered to the stream when feces were placed 2.13 m away from the stream. At the same distance with simulated frozen ground conditions, 13.7 million FCS were delivered to the stream.

Sedimentation Fecal coliforms are deposited in streams through runoff or direct deposition of fecal matter into the stream (Sherer et al. 1988). Once in the stream, FCS often become trapped in sediment and begin to die off (Larsen et al. 1994). Surviving FCS can be released back into the water column through disturbance of bank or bottom sediments along the stream course. This can occur following increased streamflow due to rain or snowmelt, or as a result of cattle wading in streams or trampling stream banks. To study the

role that cattle can play in releasing sediment-trapped FCs, Sherer et al. (1988) experimented with the trapping and release of FCs by bottom sediments in a stream in central Oregon. They found that greater than 89% of the bacteria in a manure slurry that was released into the stream appeared to settle in the stream sediments. When the stream-bottom sediments were disturbed by raking, 1.8 million–760 million FC/m^2 were released back into the water column. This represented a 17.5-fold increase in FC levels in the water column. From their findings, Sherer et al. (1988) concluded that the time cattle spend in a stream can have a significant effect on water quality.

The results of Sherer et al. (1988) were supported by other studies. Bickie et al. (1988) also experimented with releasing a manure slurry into a stream, and found that approximately 95% of the FCs settled in the bottom sediments within 50 m of the deposition site. Some additional settling occurred in the next 250 m. Gary and Adams (1985) experimented with disturbing stream-bottom sediments in a watershed in southern Wyoming, and also found that FC concentrations increased in the water column following disturbance. Fecal coliform levels increased only 1.7 times, however; much lower than the increase reported by Sherer et al. (1988). Fecal coliforms have been found to be up to 100–1000 times more numerous in bottom sediments than in overlying water (Van Donsel and Geldreich 1971).

Miner et al. (1992) found that many FCs die after becoming trapped in stream sediments. In contrast, Sherer et al. (1992) suggested that sediments allow FCs to survive for months in an aquatic environment. They found that FCs had half-lives of 11–30 days when incubated with stream sediments, which was longer than when they were incubated without sediment.

3.5.3 Protecting drinking water sources from contamination by cattle The following studies provide information on management actions that could reduce cattle impacts on drinking water sources.

Alternative water sources Based on his findings of cattle fecal deposition rates in streams and the impact they have on water quality (see Section 3.5.2), Larsen (1989) suggested that providing water away from streams, especially in areas where cattle concentrate (e.g., feedlots), could reduce fecal contamination of water sources. Miner et al. (1992) evaluated the effectiveness of an alternative water source in reducing the amount of time cattle spent in the stream in a watershed in Oregon. The study was done in a pasture where cattle ranged freely and were fed hay during the winter months. A stream ran through the length of the pasture. The pasture was fenced to create two grazing areas. In one area, cattle had access only to water in the stream. In the other area, a water tank was placed about 33 m away from the stream. Hay was placed at about equal distances from the stream in each pasture. This location was about 7 m from the water tank in the one pasture. In the pasture where the stream was the only water source, each animal spent an average of 14.5 minutes in the stream. In the pasture with the supplemental water source, each animal spent an average of 0.17 minutes in the stream. This represented almost a 99% reduction in the time cattle spent drinking and lingering in the stream. The researchers believed that cattle spent less time in the stream because the water in the tank was warmer than the stream water, and the cattle had easier access to the tank than to the stream (the water tank was placed on firm dry ground, whereas access to the stream was over steep, muddy ground). Both Larsen (1989) and Miner et al. (1992) suggest that the use of

water tanks could not only help reduce cattle fecal contamination of streams, but could also provide economic benefits to ranchers, since the cattle would expend less energy in obtaining water, and would have a constant supply of clean water.

Buffer zones There have been few studies on the role of buffer strips in controlling bacterial concentrations in surface runoff that apply to rangelands, and the results of those studies have not always been consistent (Larsen et al. 1994). Doyle et al. (1975) spread dairy cow manure over gravelly silt loam soils and found that forested buffer strips prevented movement of fecal bacteria beyond 3.8 m. Glenne (1984) used a model to simulate generation of water pollution in three watersheds in northern Utah and found that a 50-m buffer strip was needed to reduce bacterial concentrations by 90% on a 10% slope, while a 90-m strip was needed for a 20% slope. In contrast to these studies, Dickey and Vanderholm (1981) showed that while vegetative filters up to 400 m in length could reduce 80% of nutrients and solids in feedlot runoff, they did not significantly reduce bacterial concentrations.

In an attempt to clarify the role that buffer strips play in controlling runoff in cattle grazing areas, Larsen et al. (1994) used a simulated rainfall experiment to assess the effectiveness of vegetation buffer strips in reducing concentrations of cattle fecal bacteria in streams. Kentucky bluegrass (*Poa pratensis*) sods were used as buffer strips, and a series of runoff and infiltration trials were done with dairy cattle feces placed at 0, 0.61, 1.37, and 2.13 m from a collection point that was used to simulate a stream. The authors found that even a narrow buffer strip significantly reduced bacteria transported into the stream. After 30 minutes of simulated rainfall, the number of bacteria escaping from feces within 0.61 m of the stream was 83% less than that from feces deposited in the stream. With a buffer of 2.13 m, bacterial loads were reduced by 95%.

Vegetation cover Pasture rotation has often been advocated as a way to improve forage production on rangelands. Periods of non-use can also result in increased cover of vegetation and litter, which in turn can result in improved hydrologic conditions due to increased infiltration rates (Environmental Protection Agency 1979). Packer (1953) has suggested that 70% ground cover of vegetation and litter is needed to minimize runoff and erosion levels, but that higher levels of ground cover are needed on sites with increased levels of trampling. It is not known, however, whether these findings would apply to all situations in British Columbia. An Environmental Protection Agency (1979) report suggests that these findings may apply only to western mountain rangelands where maintenance of 70% vegetation cover is biologically possible. Such findings would not apply to arid and semi-arid rangelands where vegetation cover may be less than 70%, even on pristine areas.

Cattle management One method of controlling cattle-derived pathogen levels in drinking water sources is to reduce cattle exposure to diseases through proper calf management (Centre for Coastal Health 1996). This entails managing diarrhea outbreaks in calves (through fluid and electrolyte replacement therapy), and in reducing calf exposure to the parasite (Centre for Coastal Health 1996). Radostits et al. (1994) provide the following measures to reduce calf exposure to the *C. parvum* parasite:

- calving areas should be kept clean and dry;
- diarrheic calves should be kept separated from healthy calves;
- older calves should be separated from young, potentially susceptible calves;
- calving pens, if used, should be thoroughly cleaned between calving rounds;
- healthy calves should be attended to before sick calves to ensure that they remain healthy;
- utensils used to treat calves should be sterilized daily;
- other animals, such as dogs and cats, should be kept out of calf rearing areas; and
- fecal material should be removed from calving areas daily.

Additionally, local concentrations of *C. parvum* can be limited, and cattle exposure to the pathogen can be reduced, by:

- alternating bedding and feeding areas;
- increasing the size of the calving area if the site is wet or muddy;
- locating calving areas on sites where surface contamination will not drain into human or animal drinking water sources;
- removing feces and bedding material from calving areas so that the underlying soil is exposed; and
- using seasonal pasture rotations on rangelands (Radostits et al. 1994; Centre for Coastal Health 1996).

Recommendations for reducing the risk of *C. parvum* contamination of water on Crown rangeland within watersheds used for domestic water supplies (community watersheds) in British Columbia include deferring turn-out of cattle until scouring (diarrhea) has been cleared up and prohibiting calving on Crown rangeland within community watersheds (D. Fraser, pers. comm.).

3.6 Other Sources of Waterborne Diseases

3.6.1 Humans, wildlife, and *Cryptosporidium parvum* Watershed management practices can significantly affect *C. parvum* concentrations in surface waters. Throughout North America, *C. parvum* levels have been 2.3–50 times higher in waters subjected to sewage pollution, human recreation, or agricultural, industrial, or forestry activities than in waters from protected watersheds (i.e., those with restricted human and livestock activity) (Rose 1988; Hansen and Ongerth 1991; LeChevallier et al. 1991; Rose et al. 1991).

Waters regarded as pristine, however, are not necessarily free of *C. parvum* contamination. In a survey of surface and ground water in 17 U.S. states, 39% (n=100) of all pristine water sources tested contained *C. parvum* oocysts (Rose et al. 1991). Water samples were considered pristine if they were taken from areas without agricultural activities or sewage treatment facilities, and had limited human access. In such areas, wildlife may have been responsible for the background levels of oocysts detected. In western Washington, Hansen and Ongerth (1991) attributed the original source of *C. parvum* oocysts in waters from both protected and unprotected watersheds to wildlife in upland areas of the watersheds. Lucas (1998) reported concentrations of both *G. lamblia* and *C. parvum* in the Nitinat River and San Juan River watersheds of British Columbia even though the major activity in the areas was forestry, and human populations were low. *C. parvum* has been isolated from wild mammals such as beaver, coyote, raccoon, black bear, and mule deer (Table 1) (Atwill 1996; Centre for Coastal Health 1996).

3.6.2 Humans, wildlife, and *Giardia lamblia* *G. lamblia* contamination can occur through discharges of sewage or industrial effluent into drinking water supplies or from animal or human activity in community watersheds. In 95 outbreaks of waterborne giardiasis in the United States, 71% resulted from sewage contamination of surface waters, and another 12% resulted from contaminated groundwater (Wallis 1994). In a study of surface water treatment plants in the United States and Alberta, LeChevallier et al. (1991) found the highest concentrations of *G. lamblia* in source waters that had been contaminated with sewage or industrial effluents. *G. lamblia* levels in this study were also negatively correlated with levels of watershed protection. Water polluted with urban effluent had *G. lamblia* cyst concentrations 10 times higher than water from protected watersheds (LeChevallier et al. 1991).

Protecting water sources from effluent contamination does not necessarily ensure clean drinking water supplies. Between 1965 and 1984, contaminated water supplies were the source of 69% of the outbreaks of waterborne giardiasis in the United States (Craun and Jakubowski 1987). Many of these water supplies came from mountain streams or lakes that were not contaminated with sewage or wastewater discharges (Craun and Jakubowski 1987). Human and/or animal activity are probable sources of *G. lamblia* contamination of water in such areas. *G. lamblia* has been isolated from at least 40 species of animals (Kulda and Nohynkova 1978), but humans, beavers, muskrats, domestic dogs, and cattle are considered to be the most common source of *G. lamblia* contamination of drinking water (Wallis 1994). Determining the potential for infectivity of humans from animal sources has been difficult, but there is evidence to suggest that *G. lamblia* can be transmitted to humans from beavers, muskrats, mule deer, domestic dogs, domestic cats, and sheep (Davies and Hibler 1979; Wallis 1994). Isaac-Renton et al. (1993) suggest that aquatic mammals have the potential to contribute significantly to the spread of giardiasis to humans. Several studies have shown infection rates to be as high as 15% in beavers, and up to 95% in muskrats (Jakubowski 1990). These species are also known to be large reservoirs for *G. lamblia*. For example, beavers can shed 100 million cysts/animal/day (Monzingo 1985), and muskrats can shed up to 30 million cysts/animal/day (Wegrzyn 1988). In British Columbia, there have been 13 outbreaks of giardiasis since 1980 (B.C. Ministry of Health 1997). The potential source of the outbreak was identified in five of these cases. Beavers were implicated in three outbreaks, while an unidentified wildlife source was suspected in the other two (B.C. Ministry of Health 1997).

There is also evidence that *G. lamblia* can be transmitted from humans to beavers, muskrats, and domestic dogs and cats (Wallis 1994). Wildlife exposure to human sources of *G. lamblia* may have at least partially explained the results of one study. Ongerth et al. (1995) compared *G. lamblia* cyst levels in two watersheds in the Olympic Mountains in Washington State. One watershed received more than 10 times the recreational use than the other watershed. Although there was no significant difference in cyst concentrations in water samples between the two watersheds, infection levels in mammals were higher in the watershed that had greater human use. This suggests that mammals in that watershed may have been exposed to human-derived cysts. Analysis of data from the two sites combined indicated that cyst concentrations in water samples were significantly positively correlated with human use levels and with cyst prevalence in mammals.

4 CASE HISTORIES

Case histories of waterborne disease outbreaks in British Columbia were obtained primarily from reports and documents produced by the B.C. Ministry of Health, the East Kootenay Community Health Services Society, and the B.C. Ministry of Environment, Lands and Parks. Additional information was provided by the B.C. Ministry of Forests and the B.C. Ministry of Agriculture, Fisheries and Food.

4.1 Cryptosporidiosis

Between 1980 and 1997, there were two recognized outbreaks of cryptosporidiosis in British Columbia—one in Cranbrook and one in Kelowna. Cattle were implicated in both of those outbreaks (B.C. Ministry of Health 1997). Cryptosporidiosis has been a reportable disease for humans only since 1995 (Centre for Coastal Health 1996); thus, there may have been earlier unrecognized outbreaks.

4.1.1 Cranbrook 1996

British Columbia Ministry of Health report The following account of the Cranbrook outbreak is summarized from the B.C. Ministry of Health's 1996 report.

Between June 19 and July 12, 1996 136 cases (29 lab-confirmed, 107 clinical) of cryptosporidiosis were identified in the Cranbrook area. Based on a survey of household members who experienced disease symptoms during that time, and whose residence was supplied with water from the city's water system, the estimated number of infected individuals was 2097. Twenty-eight variables were tested for association with confirmed cases of the disease. Of these, consumption of unfiltered city water was most strongly associated with incidences of the disease. People who had diarrhea also drank more than twice the volume of city water (prior to becoming ill) than people who were not ill.

The main sources of Cranbrook's drinking water supply are Joseph Creek and a diversion from Gold Creek, both of which flow into Phillips Reservoir. On May 18, 1996 167 cows and 167 calves were turned out on the grazing unit near the reservoir and sections of Joseph and Gold Creeks. The reservoir and 200 m of Joseph Creek upstream of the reservoir are fenced to prevent cattle access. Cattle were observed grazing upstream of the fenced area, and on 23 June 1996, cattle feces were found immediately upstream of the fence. Manure-contaminated material was removed from up to 30 m on each bank of Joseph Creek and the Gold Creek diversion, for a distance of about 5.5 km. In total, 2091 litres of fecal material was collected.

Suitable deer, muskrat, and beaver habitat existed around the reservoir. A beaver dam and lodge were present, but did not appear to be used recently. A local rancher reported 123 elk in the watershed, and collected two samples of deer feces for testing.

Other possible sources of contamination of the city's water supply included two watermain breaks, and flow testing of fire hydrants from mid-May to June 20, 1996. Customer complaints rose during the fire hydrant testing, indicating that sediments had been disturbed and were being distributed through the water system. Neither of these incidences, however, were considered by the Ministry of Health to be related to the outbreak of the disease.

Twenty-nine people, an unspecified number of raw water samples, and seven of eight cattle feces samples tested positive for *C. parvum* oocysts. The morphological characteristics of these oocysts were consistent with *C. parvum*.[2] This suggested to the authors of the B.C. Ministry of Health report that cattle were a possible source of contamination at the Phillips Reservoir. It is not known, however, if the cattle feces tested were from one animal or several, or whether cattle became infected before or after entering the area.

Subsequent commentary There is no indication in the report that fecal samples from wild mammals or human recreationists in the watershed were tested for presence of the parasite. A later East Kootenay Community Health Services Society memorandum (Arsenault 1997), however, indicates that one sample of deer feces collected from the watershed on July 10, 1996 was negative, one sample of elk feces collected on nearby rangeland on September 10, 1996 was negative, and one sample of horse feces collected from the watershed on September 16, 1996 was positive. Both the B.C. Ministry of Health report and the memorandum note that there may have been other sources of *C. parvum* contamination in the watershed, but that cattle could have acted as the main amplification host because they were the major source of feces around the water source. M. Wetzstein (Health Management Veterinarian, B.C. Ministry of Agriculture, Fisheries and Food) questioned whether cattle could have contributed large enough loads of oocysts to cause a disease outbreak of the magnitude reported, given the timing of cattle turn-out. Wetzstein notes that cattle were released into the watershed on May 18, only 3 days before the start of the "epidemic curve" recorded by the B.C. Ministry of Health. He also notes that very few cattle were located above the reservoir by May 21. G. Arsenault (Medical Health Officer, East Kootenay Community Health Services Society) comments that differentiating the case that marks the onset of an epidemic from the case that is sporadic background can be difficult. The earliest one or two cases may or may not have been part of the outbreak.

Arsenault (1997) states that no feces from wild animals were found around the reservoir at the time of the outbreak. There is no indication whether surveys for muskrats or beavers were made during this time, only that an existing beaver lodge and dam appeared uninhabited. According to G. Griffin (B.C. Ministry of Forests, Cranbrook Forest District) there is substantial beaver activity in the watershed. In years with high runoff levels, beaver dams in the watershed are often breached (G. Griffin, pers. comm.). This, in turn, can lead to scouring of pond bottom sediments and the potential release into the water of pathogens trapped in those sediments. Runoff levels may have been high enough in 1996 to cause such breaching of the beaver dam near the reservoir (G. Griffin, pers. comm.).

The B.C. Ministry of Health report states that "rainfall and runoff from snow melt during this time period (May 19 to June 9, 1996) may have altered flow patterns and thermal stratification in the reservoir, resulting in 'short circuiting' of the normal sedimentation activities of the reservoir." In a letter responding to the B.C. Ministry of Health report, L. McDonald (Impact Assessment Biologist, B.C. Ministry of Environment, Lands and Parks, Kootenay Region) says that the B.C. Ministry of Health statement about rainfall and runoff suggests that "...1996 was an unusual year which contributed to

2 Subsequent testing of these isolates identified them as *C. parvum* Genotype 2, a genotype previously isolated from human, cattle, sheep, goat, and deer hosts (Ong et al. 1999, Ong et al. 2002).

the conditions that lead to an outbreak of *C. parvum*." McDonald (1997) presents rainfall records from Environment Canada (1996) for the Cranbrook Airport indicating that precipitation in May 1996 was 86% higher than the 1968–1995 average, although June 1996 rainfall was 17% lower than the 27-year average. A report by Lucey and Barraclough (1997) states that flooding occurred in the upper watershed in early June 1996 due to a "rain-on-snow event" and that a 13.5-m section of the Gold Creek reservoir dam washed out on May 18, 1996.

McDonald (1997) also raises the point that cattle have grazed in this watershed for years, and asks why a *C. parvum* outbreak attributable to cattle had not occurred in this watershed prior to 1996. In reply to McDonald's comment, Arsenault (1997) states that, historically, cattle were not turned out in the watershed until later in the year. Arsenault points out that at later times, calves would be older and therefore more resistant to the disease, and thus would be less likely to shed large amounts of infective oocysts. Arsenault does not say, however, how much later the cows would have traditionally been turned out, or whether, in fact, this would have been outside the main period of infectivity in calves. It is known, however, that the main period of infectivity in calves is from age 3–35 days (Aiello 1998). In a subsequent communication, the issue of the timing of cattle turn-out in the watershed was questioned by G. Griffin. According to Griffin (pers. comm.), cows have traditionally been turned out in the area around the middle of May and often in greater numbers than in 1996. M. Wetzstein (pers. comm.) also questioned why a *C. parvum* outbreak had not occurred before 1996. Wetzstein noted that Cranbrook's drinking water supply originally came from a creek that ran through a cattle holding area. Presumably, there was potential in the past for a *C. parvum* outbreak due to cattle in the watershed, yet none was recorded. Arsenault (pers. comm.) notes that the province did not have the ability to test for *C. parvum* in stool samples until about 1983 and that cryptosporidiosis became a reportable disease for humans only in 1995 (verified by the Centre for Disease Control, Parisitology Section). Arsenault also points out that cryptosporidiosis causes rapid and short-lived diarrhea in most cases. Individuals with these symptoms are not likely to be tested for cryptosporidiosis unless massive outbreaks occur.

McDonald (1997) also notes that, despite the evidence supporting the role of cattle in the Cranbrook *C. parvum* outbreak, it is not known if cattle-derived oocysts were, in fact, viable. This point was also made by T. Johnstone (epidemiologist) in a letter to the City of Cranbrook regarding the *C. parvum* outbreak. Johnstone (1996) notes that *C. parvum* oocysts occur in two forms: one of which is believed to not survive outside the host; the other of which can survive for many weeks in the environment, and which is the main mode of transmission of the parasite.

The B.C. Ministry of Health report states that the watershed is open to public recreation such as hunting, fishing, camping, and boating. According to Griffin (pers. comm.), the watershed receives substantial recreational use. Active logging roads and trails provide easy access to much of the watershed. As a result of human activity, garbage must be removed periodically from the watershed. Types of garbage removed from the creeks and the area around the reservoir include baby diapers, domestic animal carcasses, and wild animal carcasses left by hunters (G. Griffin, pers. comm.), although items of this description were not found when the area was inspected shortly after the outbreak. The B.C. Ministry of Health report did not indicate whether

human activities in the watershed could have contributed to the presence of waterborne *C. parvum*.

Both Johnstone (1996) and McDonald (1997) questioned whether the disease outbreak was actually associated with the drinking water supply. Johnstone (1996) acknowledges that although it is biologically plausible that water may have been involved in the outbreak, a positive statistical correlation between drinking water and disease cases implies only an association between the two variables, not that contaminated water caused the outbreak. However, Arsenault (1997) states that the Cranbrook outbreak met all the usual epidemiological criteria for identification of waterborne outbreaks. People with diarrhea were spread throughout the community, no other possible source of infection was identified, and people with diarrhea were 9 times more likely to have drunk unfiltered city water prior to the onset of illness than had people without diarrhea. Furthermore, there was less than a one in 200 chance that the correlation between water consumption and diarrhea cases was due simply to chance.

McDonald (1997) also questions the association between disease cases and the amount of city water consumed. McDonald asks: "Did the group with diarrhea contract the disease because they drank more water than others, or did they drink more water because they had diarrhea and were dehydrated?" Arsenault (1997) states that people with diarrhea drank more water before they became ill than people who did not become ill. This point was not made in the original B.C. Ministry of Health (1996) report. McDonald (1997) also notes that the association between disease cases and amount of drinking water consumed assumes that the quantity of oocysts was uniform over time and throughout the water sources. Arsenault (1997) agrees that oocysts do not have a uniform distribution in water supplies, but points out that over time, in a large group of people who drink contaminated water, those who drink more water will, on average, be expected to ingest proportionally more oocysts than those who drink less water. McDonald also stresses that if there was a dose response between oocyst consumption and cryptosporidiosis, it would have been reasonable to expect a rise in disease cases after the beaver dam on Joseph Creek was removed on June 26, 1996, since many people ignored the "boil water advisory" that was issued on June 21. McDonald (1997) notes that after the beaver dam was removed, the oocyst count in the water rose to 1.6 million oocysts/100 L.[3] This, according to McDonald (1997), was one of the highest counts ever recorded in North America. Arsenault (1997) agrees that oocyst counts in the drinking water supply before the outbreak were unknown, but cites Meinhardt et al. (1996) in saying that water sampling is not a reliable way to estimate risk to people drinking the water. This is because water sampling measures oocyst counts, and does not distinguish between viable infective cysts and dead cysts. Furthermore, because viability drops off with time (older cysts are less viable than younger cysts), cysts released from disturbed sediments are less likely to be infective because they tend to be older.

Johnstone (1996) notes that although the Cranbrook incidence was the first reported outbreak of cryptosporidiosis associated with drinking water

3 There is disagreement regarding the sequence of these events. Don Corrigal (East Kootenay Community Health Services Society) points out that information provided by the City of Cranbrook lists the date of removal of the beaver dam and lodge as June 26, 1996. The test sample that produced the 1.6 million oocysts/100 L count was taken on June 25, 1996; therefore, the removal of the beaver dam could not have contributed to the high oocyst count.

in British Columbia, the background rates of human cryptosporidiosis in different parts of British Columbia are unknown. According to Johnstone (1996), before the cryptosporidiosis outbreak in Milwaukee, Wisconsin in 1993, the background rate of watery diarrhea in humans was 0.5%. If a similar rate was applied to the population of 18 000 in Cranbrook, 90 people would be expected to have diarrhea in any month (Johnstone 1996). Arsenault (pers. comm.) notes that this number reflects expected background incidence of diarrhea and should not be compared with the background incidence of reported cases, which is far lower. Only a fraction of reportable acute diarrheal illnesses are actually reported because:

1. most individuals with acute diarrhea do not go to the doctor;
2. those who do, do not usually get stool tests ordered;
3. of those who do have stool tests ordered, not all will submit a sample, especially when their symptoms abate;
4. even if a sample is submitted by an individual with cryptosporidiosis, not all will test positive (false-negative test results). at most, the stool test used is only positive one-third of the time in individuals with cryptosporidiosis; and
5. individuals who are exposed to *C. parvum* over a long period of time develop partial immunity, and therefore do not develop symptoms as severe as those who do not have this immunity.

It should be noted that of the 136 cases reported in the Cranbrook outbreak, 107 cases (i.e., the clinical cases) showed symptoms of infection, but laboratory tests failed to identify the causative agent of those symptoms (B.C. Ministry of Health 1996). McDonald (1997) observes that there was a 21% chance (29 out of 136) that *C. parvum* was the cause of the reported diarrhea cases, but there was also a 79% chance (107 out of 136) that it was not. Arsenault (1997), however, states that not all fecal samples from a person with cryptosporidiosis will test positive for the parasite and that the 21% positivity rate found in Cranbrook is similar to rates reported for large urban outbreaks of the disease. In the Milwaukee outbreak (for example), only about 30% of all diarrhea stools tested contained oocysts (Mackenzie et al. 1994).

4.1.2 Kelowna 1996 An outbreak of cryptosporidiosis also occurred in 1996 in Kelowna. There were 157 confirmed cases of the disease, but the estimate of the actual number infected was 10 000. Contamination from urban, rural, and agricultural sources was suspected to be responsible for the outbreak (B.C. Ministry of Health 1997). The suspected source of agricultural contamination was runoff from cattle feedlot operations. The Kelowna watershed is so large that the actual sources of contamination could not be determined, nor the degree to which they contributed to the outbreak (B. Boettger, pers. comm.).

4.2 *Campylobacter*

Only one outbreak of *Campylobacter* in British Columbia was attributed to cattle, but that link was tenuous. The outbreak occurred at the ski hill in Fernie in 1993. Thirty-five cases of the disease were confirmed in the local human population. The ski lodge drew their water from a nearby creek, but did not use their chlorination system due to customer complaints about the taste of chlorine in the drinking water. Cattle were implicated in the outbreak

because a local livestock operation was known to graze their cows upstream of the lodge's intake system. It is not known, however, if the cattle were tested for the presence of *Campylobacter* (B. Boettger, pers. comm.). Regardless of the source of the disease, the outbreak likely could have been prevented if the lodge had used its chlorination system, since chlorine can effectively control bacterial organisms such as *Campylobacter*.

5 SUMMARY AND INFORMATION NEEDS

This section is based on the results of the literature review, case histories, and interviews. The research needs identified are extracted from a list compiled from interviewee responses (Appendix 1).

5.1 Livestock Use and the Incidence of Cryptosporidiosis and Giardiasis in Humans

There were no scientific studies found that examined the connection between livestock use in watersheds and the incidence of waterborne cryptosporidiosis or giardiasis in humans. A few studies examined the correlation between livestock use and the presence of oocysts/cysts in surface water (Section 3.1.2, Section 3.1.3). Despite wide variability and conflicting results within this group of studies, there seems to be a sufficient correlation between increased levels of oocysts/cysts in water and the presence of cattle to justify further examination of cattle as a source of oocysts/cysts in surface water. It should be noted, however, that the strongest correlations were with intensively managed cattle operations (e.g., dairy farms, overwintering areas). There was less evidence supporting a correlation between increased oocysts/cysts in surface water and cattle management on rangeland.

No scientific literature was located verifying that cattle-derived oocysts/cysts in surface water indicate a health risk of cryptosporidiosis or giardiasis to humans. One study reported that calf-derived oocysts, fed directly to humans, could lead to cryptosporidiosis. Another study demonstrated that many human cryptosporidial infections in British Columbia were due to a genotype of *C. parvum* that is known to be transmissible from cattle to humans, thereby suggesting that cattle are a possible source. However, this study does not rule out humans, sheep, goats, and deer as possible sources. There was agreement in the literature that the inability to determine oocyst/cyst survivability, potential infectivity, pathogenicity, or virulence complicates efforts to link livestock with waterborne outbreaks of cryptosporidiosis or giardiasis.

Interview respondents were not in agreement as to whether livestock contributed to the incidence of waterborne disease in humans (Appendix 1). Those who believed that livestock contributed to waterborne disease in humans often cited the Cranbrook outbreak of cryptosporidiosis as the main evidence for their belief. Other respondents noted that there are many potential sources of contamination and that water supplies derived from watersheds without livestock have been found to contain *C. parvum* and *G. lamblia* oocysts/cysts. This belief is supported by studies confirming the presence of *C. parvum* and *G. lamblia* in a number of animal species in British Columbia (Section 3.6). Some respondents noted that simply finding oocysts/cysts in surface waters where livestock are present did not necessarily indicate a threat to humans.

Summary

- There is some scientific evidence that cattle concentrations lead to increased numbers of oocysts/cysts in surface water.
- There is no scientific evidence that cattle-derived oocysts/cysts in surface water can survive and infect humans at a pathogenic level. There is evidence that calf-derived oocysts, fed directly to humans, can lead to cryptosporidiosis. There is evidence that many human cryptosporidiosis cases in British Columbia are of the *C. parvum* genotype that is known to be transmissible from cattle.
- The Cranbrook outbreak of cryptosporidiosis (Section 4.1.1) provides one of the few convincing indications that there may be a link between cattle and the incidence of cryptosporidiosis in humans in British Columbia.

Research needs identified by interviewees

- Ecology of waterborne pathogens, including their movement, viability, and survivability from the host source through the entire water system to the tap;
- Concentrations of waterborne *C. parvum* necessary to create outbreaks in the human population; and
- Prevalence of diseases in cattle herds throughout British Columbia.

5.2 Management Actions with Potential to Reduce or Eliminate the Potential Risk of Human Cryptosporidiosis and Giardiasis Linked to Livestock Use

There is a lack of studies with useful quantitative information concerning livestock management techniques that can reduce the potential risk of human cryptosporidiosis and giardiasis. Except for a study by Ong et al. (1996), few authors have provided quantitative information useful for watershed management. Details such as cattle numbers, season of use, and age class of the animals are seldom included. Many of these studies were designed to detect if cattle/livestock could be correlated with oocyst/cyst presence, not to understand the mechanism by which this occurred. As a result, the only management recommendations possible from these kinds of studies would be to exclude or retain livestock in the watershed.

There is a fairly large body of literature on management techniques for reducing fecal coliform contamination of surface water (Section 3.5). Although FCs are generally considered to be poor indicators of protozoan pathogens such as *C. parvum* and *G. lamblia*, they do indicate that fecal contamination has occurred (a necessary factor for *C. parvum* and *G. lamblia* occurrence in surface water). This currently provides the most useful quantitative information concerning livestock management techniques with the potential to reduce the risk of human cryptosporidiosis and giardiasis.

Many interview respondents suggested that cattle access should be restricted in community watersheds. Respondents also suggested a number of management techniques to reduce the risk of waterborne diseases linked to cattle (Appendix 1). A few respondents stated that present provincial guidelines for livestock use of community watersheds should be properly implemented.

Summary

- Quantitative studies that may be useful for the management of livestock in community watersheds to reduce cryptosporidiosis and giardiasis are not available.

- Studies on fecal coliform contamination provide the most useful quantitative information concerning livestock management techniques with the potential to reduce the risk of human cryptosporidiosis and giardiasis.
- Some individuals believe that cattle access should be limited in community watersheds.

Research needs identified by interviewees

- Associations between cattle management practices and outbreaks of waterborne diseases;
- Age at which calves develop some resistance to *C. parvum*;
- Impacts of cattle in riparian areas in local watersheds (e.g., rates of manure deposition in and near streams, and the impact on water quality); and
- Methods to reduce impacts of cattle in riparian areas (e.g., use of alternative watering systems).

5.3 Potential Sources of *Cryptosporidium parvum* and *Giardia lamblia* other than Livestock

There is a reasonable body of scientific evidence showing that many wildlife species carry *C. parvum* and *G. lamblia* (Section 3.6). *C. parvum* has been isolated from wild mammals such as beaver, coyote, raccoon, black bear, and mule deer (Table 1). *G. lamblia* has been isolated from at least 40 species of animals including beaver, muskrat, and domestic dog (Section 3.6.2). Many interview respondents also cited human fecal contamination as an important potential source of *C. parvum* and *G. lamblia* in community water supplies.

Summary

- Wildlife and human fecal contamination are important potential sources of *C. parvum* and *G. lamblia* in community water supplies.

Research needs identified by interviewees

- Sources of waterborne pathogens, and their significance in outbreaks of waterborne diseases (one way to accomplish this may be through the use of genetic markers); and
- Prevalence of diseases in different wildlife species throughout British Columbia.

6 FUTURE RESEARCH

Future research on the cattle/waterborne disease issue should be designed with the objective of producing information useful for integrated watershed management. For example, more studies are required that examine associations between cattle management practices and the occurrence of *Giardia* cysts/*Cryptosporidium* oocysts in water. Studies are also required that focus on mechanisms, such as determining how viable oocysts/cysts move from manure to surface water to the tap. Basic quantitative information on this topic is lacking. Future cattle/waterborne disease studies should include as much quantitative information on cattle management in the affected watershed as possible. The minimum information required to quantify the cattle management system is:

- the density of cattle in an area where they have a potential to contribute to oocyst/cyst loads in surface water;
- the amount of time that cattle spend in an area where they have a potential to contribute to oocyst/cyst loads in surface water;
- the ages of the cattle, especially whether calves younger than 3 months old are present;
- the prevalence of cryptosporidiosis/giardiasis in the herd; and
- the cattle management system employed (e.g., dairy or beef, intensively pastured or free range, type of pasture rotation, season of use).

7 CONCLUSIONS

The debate surrounding the 1996 outbreak of cryptosporidiosis in Cranbrook (see Section 4) illustrates the degree of disagreement surrounding the role that cattle play in waterborne disease outbreaks in British Columbia. The resolution of this issue will be difficult because of a lack of directly applicable scientific evidence, and inadequate water sampling methodology.

In theory, present provincial regulations governing livestock in community watersheds provide adequate guidance to protect against waterborne disease outbreaks originating from livestock. Managers responsible for implementing these regulations, however, are faced with limited quantitative biological information on which to base their decisions. Indeed, very little scientific information has been identified that directly addresses livestock management and cryptosporidiosis/giardiasis in humans. The literature on management techniques for reducing fecal coliform contamination of surface water by livestock currently provides the most useful quantitative information concerning livestock management techniques with the potential to reduce the risk of human cryptosporidiosis and giardiasis.

REFERENCES

Aiello, S.E. (editor). 1998. The Merck veterinary manual. 8th ed. Merck and Co., Whitehouse Station, N. J.

Alderfer, R.B. and R.R. Robinson. 1947. Runoff from pastures in relation to grazing intensity and soil compaction. J. Amer. Soc. Agron. 39:948–58.

Aldom, J.E. and A.H. Chagla. 1995. Recovery of *Cryptosporidium* oocysts from water by a membrane filter dissolution method. Lett. Appl. Microbiol. 20:186–7.

American Water Works Association. 1994. Preventing waterborne disease: is your system at risk? Satellite teleconference participant guide. Denver, Colo.

Arsenault, G. 1997. Memorandum to Les McDonald, B.C. Ministry of Environment, Lands and Parks, Cranbrook, B.C. East Kootenay Community Health Services Society, Cranbrook, B.C. Unpublished.

Atwill, E.R. 1996. Assessing the link between rangeland cattle and waterborne *Cryptosporidium parvum* infection in humans. Rangelands 18:48–51.

Barer, M.R. and A.E. Wright. 1990. A review: *Cryptosporidium* and water. Letters in Appl. Microbiol. 11:271–7.

Baxter-Potter, W. and M.W. Gilliland. 1988. Bacterial pollution in runoff from agricultural lands. J. Environ. Qual. 17:27–34.

Bell, A., R. Guasparini, D. Meeds, R.G. Mathias, and J.D. Farley. 1993. A swimming pool-associated outbreak of cryptosporidiosis in British Columbia. Can. J. Public Health 84:344–7.

Berry, T., T. Webb, and C. Perrin. 1993. Forestry–drinking water quality interactions: final workshop report. ESSA Environmental and Social Systems Analysts Ltd., Vancouver, B.C.

Bickie, H.A., B.M. Sherer, J.A. Moore, J.R. Miner, and J.C. Buckhouse. 1988. Fate of organisms from manure point loading into rangeland streams. Amer. Soc. Agric. Engineers, St. Joseph, Mich. Paper No. 88-2081.

British Columbia Ministry of Environment, Lands and Parks. Forest and range management impacts on water quality. Bibliography. April 24, 1998. <www.elp.gov.bc.ca/wat/wq/farm/farma.html>

British Columbia Ministry of Forests. 1997. Application of the community watershed guidebook from a range management perspective. Victoria, B.C. Unpublished.

British Columbia Ministry of Forests and British Columbia Ministry of Environment, Lands and Parks. 1996. Community watershed guidebook. Forest Practices Code of British Columbia. Victoria, B.C.

British Columbia Ministry of Health. 1982. British Columbia drinking water quality. Victoria, B.C.

_____. 1996. Outbreak of cryptosporidiosis associated with municipal drinking water: Cranbrook, British Columbia, June 1996. Report to the Cranbrook Local Board of Health. East Kootenay Health Unit, Cranbrook, B.C.

_____. 1997. Waterborne disease outbreaks in British Columbia 1980–1997. Victoria, B.C.

British Columbia Ministry of Municipal Affairs and Housing. 1997. Municipal water treatment plants in British Columbia. Municipal Engineering Services Memorandum. Victoria, B.C.

Bruce-Grey-Owen Sound Health Unit. 2000. The investigative report of the Walkerton outbreak of waterborne gastroenteritis. May–June, 2000. Prepared by the Bruce-Grey-Owen Sound Health Unit, Health Canada and the Ontario Ministry of Long-Term Care, Owen Sound, Ont.

Buret, A., N. den Hollander, P.M. Wallis, D. Befus, and M.E. Olson. 1990. Zoonotic potential of giardiasis in domestic ruminants. J. Infect. Dis. 162:231–7.

Casemore, D.P., S.E. Wright, and R.L. Coop. 1997. Cryptosporidiosis—human and animal epidemiology. *In Cryptosporidium* and cryptosporidiosis. R. Fayer (editor). CRC Press, Boca Raton, Fla., pp. 65–92.

Centre for Coastal Health. 1996. Summary report of the key aspects of *Cryptosporidium parvum* with special reference to cattle. Dept. of Health Care and Epidemiology, Univ. B.C., Vancouver, B.C.

Craun, G.F. 1984. Waterborne outbreaks of giardiasis. Current status. *In Giardia* and giardiasis. S.L. Erlandsen and E.A. Meyer (editors). Plenum Press, New York, N.Y. pp. 243–61.

Craun, G.F. and W. Jakubowski. 1987. Status of waterborne giardiasis outbreaks and monitoring methods. *In* Proc. international symp. on water-related health issues. C.L. Tate, Jr. (editor). Amer. Water Res. Assoc., Bethesda, Md. TPS87-3, Cited in W. Jakubowski. 1990. The control of *Giardia* in water supplies. *In* Giardiasis. E.A. Meyer (editor). Elsevier Science Publ., New York, N.Y., pp. 335–53.

Crockett, C.S. and C.N. Haas. 1997. Understanding protozoa in your watershed. J. Amer. Water Works Assoc. 89:62–73.

Cully, M.J. 1938. Grazing habits of range cattle. J. For. 36:715–7. Cited in R.E. Larsen. 1989. Water quality impacts of free ranging cattle in semi-arid environments. M.Sc. thesis. Oreg. State Univ., Corvallis, Oreg.

Current, W.L. and L.S. Garcia. 1991. Cryptosporidiosis. Clinical Microbiol. Rev. 4(3):325–58 Cited in Centre for Coastal Health. 1996. Summary report of the key aspects of *Cryptosporidium parvum* with special reference to cattle. Dept. of Health Care and Epidemiology, Univ. B.C., Vancouver, B.C.

Davies, R.B. and C.P. Hibler. 1979. Animal reservoirs and cross-species transmission of *Giardia*. *In* Waterborne transmission of giardiasis. W. Jakubowski and J.C. Hoff (editors). Proc. symp. U.S. Environ. Protection Agency, Cincinnati, Ohio, pp. 104–126. Cited in C.P. Hibler and C.M. Hancock. 1990. Waterborne giardiasis. *In* Drinking water microbiology: progress and recent developments. G.A. McFeters (editor). Springer-Verlag, New York, N.Y., pp. 271–93.

Dickey, E.C. and D.H. Vanderholm. 1981. Vegetative filter treatment of livestock runoff. J. Environ. Qual. 10:279–84. Cited in R.E. Larsen, J.R. Miner, J.C. Buckhouse, and J.A. Moore. 1994. Water-quality benefits of having cattle manure deposited away from streams. Bioresource Technology 48:113–8.

Doran, J.W. and D.M. Linn. 1979. Bacteriological quality of runoff water from pastureland. Appl. and Environ. Microbiol. 37:985–91.

Doyle, R.C., D.C. Wolf, and D.F. Bezdicek. 1975. Effectiveness of forest buffer strips in improving the water quality of manure polluted runoff. *In* Managing livestock wastes. Proc. 3rd International symp. on livestock wastes. Amer. Soc. Agric. Engineers St. Joseph, Mich., Publ. Proc-275, pp. 299–302. Cited in R.E. Larsen, J.R. Miner, J.C. Buckhouse, and J.A. Moore. 1994. Water-quality benefits of having cattle manure deposited away from streams. Bioresource Technology 48:113–8.

Dupont, H.L., C.L. Chappell, C.R. Sterling, P.C. Okhuysen, J.B. Rose, and W. Jakubowski. 1995. The infectivity of *Cryptosporidium parvum* in healthy volunteers. N. Engl. J. Med. 332:855–9. Cited in E.R. Atwill. 1996. Assessing the link between rangeland cattle and waterborne *Cryptosporidium parvum* infection in humans. Rangelands 18:48–51.

Environment Canada. 1996. Historical precipitation records from Cranbrook airport 1968–1996. Mountain Weather Office, Kelowna, B.C. Cited in L. McDonald. 1997. Memorandum to Julia Beatty Spence. B.C. Min. Environ., Lands and Parks, Cranbrook, B.C. Unpublished.

Environmental Protection Agency. 1979. Livestock grazing and water quality protection. Seattle, Wash.

_____. 1994. National primary drinking water regulations: enhanced surface water treatment requirements. Fed. Register 59:38832–58. Cited in E.R. Atwill. 1996. Assessing the link between rangeland cattle and waterborne *Cryptosporidium parvum* infection in humans. Rangelands 18:48–51.

Fogel, D., J. Isaac-Renton, R. Guasparini, W. Moorehead, and J. Ongerth. 1993. Removing *Giardia* and *Cryptosporidium* by slow sand filtration. J. Amer. Water Works Assoc. Nov. 1993:79–84.

Fraser Valley Health Region. 1998. The 1998 Chilliwack *Cryptosporidium* outbreak—an update. Abbotsford, B.C. Unpublished memo.

Gary, H.L. and J.C. Adams. 1985. Indicator bacteria in water and stream sediments near the Snowy Range in southern Wyoming. Water, Air, Soil Pollution 25:133–44. Cited in B.M. Sherer, J.R. Miner, J.A. Moore, and J.C. Buckhouse. 1992. Indicator bacterial survival in stream sediments. J. Environ. Qual. 21:591–5.

Gary, H.L., S.R. Johnson, and S.L. Ponce. 1983. Cattle grazing impact on surface water quality in a Colorado Front Range stream. J. Soil and Water Cons. 38:124–8.

Geldreich, E.E. 1976. Fecal coliform and fecal streptococcus density relationships in waste discharges and receiving waters. CRC Crit. Rev. Environ. Control 6 (Oct.):349–69. Cited in W. Baxter-Potter and M.W. Gilliland. 1988. Bacterial pollution in runoff from agricultural lands. J. Environ. Qual. 17:27–34.

Glenne, B. 1984. Simulation of water pollution generation and abatement on suburban watersheds. Water Research Bull. 20(2):211–7. Cited in R.E. Larsen, J.R. Miner, J.C. Buckhouse, and J.A. Moore. 1994. Water-quality benefits of having cattle manure deposited away from streams. Bioresource Technology 48:113–8.

Hansen, J.S. and J.E. Ongerth. 1991. Effects of time and watershed characteristics on the concentration of *Cryptosporidium* oocysts in river water. Appl. and Environ. Microbiol. 57:2790–5.

Hibler, C.P. and C.M. Hancock. 1990. Waterborne giardiasis. *In* Drinking water microbiology: progress and recent developments. G.A. McFeters (editor). Springer-Verlag, New York, N.Y., pp. 271–93.

Isaac-Renton, J.L. 1987. Giardiasis: a review. B.C. Med. J. 29:341–4.

Isaac-Renton, J.L., C. Cordeiro, K. Sarafis, and H. Shahriari. 1993. Characterization of *Giardia duodenalis* isolates from a waterborne outbreak. J. Infect. Dis. 167:431–40.

Isaac-Renton, J.L., W. Moorehead, and A. Ross. 1996. Longitudinal studies and *Giardia* contamination in two community drinking water supplies: cyst levels, parasite viability, and health impact. Appl. and Environ. Microbiol. 62:47–54.

Jakubowski, W. 1990. The control of *Giardia* in water supplies. *In* Giardiasis. E.A. Meyer (editor). Elsevier Science Publ., New York, N.Y., pp. 335–53.

Jarroll, E.L., J.C. Hoff, and E.A. Meyer. 1984. Resistance of cysts to disinfection agents. *In Giardia* and giardiasis: biology, pathogenesis and epidemiology. S.L. Erlandsen and E.A. Meyer (editors). Plenum Press, New York, N.Y. pp. 311–28. Cited in J.L. Isaac-Renton. 1987. Giardiasis: a review. B.C. Med. J. 29:341–4.

Jawson, M.D., L.F. Elliott, K.E. Saxton, and D.H. Fortier. 1982. The effect of cattle grazing on indicator bacteria in runoff from a Pacific Northwest watershed. J. Environ. Qual. 11:621–7.

Johnstone, T. 1996. Cryptosporidiosis in Cranbrook. Report to the City of Cranbrook, B.C. Unpublished.

Kress, M. and G.F. Gifford. 1984. Fecal coliform release from cattle fecal deposits. Water Res. Bull. 20:61–6.

Kulda, J. and E. Nohynkova. 1978. Flagellates of the human intestine and of intestines of other species. *In* Parasitic protozoa II. J.P. Kreier (editor). Academic Press, New York, N.Y., pp. 1–138. Cited in W. Jakubowski. 1990. The control of *Giardia* in water supplies. pp. 335–53. *In* Giardiasis. E.A. Meyer (editor). Elsevier Science Publ., New York, N.Y.

Kunkle, S.H. 1970. Concentrations and cycles of bacterial indicators in farm surface runoff. *In* Relationship of agriculture to soil and water pollution. Proc. Cornell Univ. conf. on Agricultural Waste Management. Rochester, N.Y. January 19–21. Agricultural Waste Management Program, Cornell Univ., Ithaca, N.Y. pp. 226–32. Cited in W. Baxter-Potter and M.W. Gilliland. 1988. Bacterial pollution in runoff from agricultural lands. J. Environ. Qual. 17:27–34.

Larsen, R.E. 1989. Water quality impacts of free ranging cattle in semi-arid environments. M.Sc. thesis. Oreg. State Univ., Corvallis, Oreg.

Larsen, R.E., J.R. Miner, J.C. Buckhouse, and J.A. Moore. 1994. Water-quality benefits of having cattle manure deposited away from streams. Bioresource Technology 48:113–8.

LeChevallier, M.W. and W.D. Norton. 1995. *Giardia* and *Cryptosporidium* in raw and finished water. J. Amer. Water Works Assoc. 87:54–68.

LeChevallier, M.W., W.D. Norton, and T.B. Atherholt. 1997. Protozoa in open reservoirs. J. Amer. Water Works Assoc. 89:84–96.

LeChevallier, M.W., W.D. Norton, and R.G. Lee. 1991. Occurrence of *Giardia* and *Cryptosporidium* spp. in surface water supplies. Appl. Environ. Microbiol. 57:2610–6.

Lucas, B. 1998. Water quality inventory of the Nitinat River and San Juan River watersheds – Vancouver Island, British Columbia – 1997. B.C. Ministry of Environment, Lands and Parks, Aquatic Envelope Committee of the Common Land Inventory Base, Victoria, B.C.

Lucey, W.P. and C.L. Barraclough. 1997. City of Cranbrook drinking water supply: annual report September 1996 – August 1997. Prepared for the City of Cranbrook, B.C. by Aqua-Tex Scientific Consulting Ltd., Victoria, B.C.

MacKenzie, W.R., N.J. Hoxie, M.E. Proctor, M.S. Gradus, K.A. Blair, D.E. Peterson, J.J. Kazmierczak, D.G. Addiss, K.R. Fox, J.B. Rose, and J.P. Davis. 1994. A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public water supply. New England J. Med. 331:161–7.

Madore, M.S., J.B. Rose, C.P. Gerba, M.J. Arrowood, and C.R. Sterling. 1987. Occurrence of *Cryptosporidium* oocysts in sewage effluents and selected surface waters. J. Parasit. 73:702–5.

Mann, E.D., L.H. Sekla, G.P.S. Nayar, and C. Koschik. 1986. Infection with *Cryptosporidium* spp. in humans and cattle in Manitoba. Can. J. Vet. Res. 50:174–8.

Mawdsley, J.L., A.E. Brooks, and R.J. Merry. 1996. Movement of the protozoan pathogen *Cryptosporidium parvum* through three contrasting soil types. Biol. Fertil. Soils 21:30–6.

McDonald, L. 1997. Memorandum to Julia Beatty Spence. Contained in a letter to Andrew Hazelwood, Executive Director, B.C. Ministry of Health, Health Protection and Safety Division, from Dennis McDonald, Regional Director of Environment and Lands, Kootenay Region, March 10, 1997. Unpublished.

Meinhardt, P.L., D.P. Casemore, and K.B. Miller. 1996. Epidemiologic aspects of human cryptosporidiosis and the role of waterborne transmission. Epidemiologic Rev. 18: 118–36.

Miner, J.R., J.C. Buckhouse, and J.A. Moore. 1992. Will a water trough reduce the amount of time hay-fed livestock spend in the stream? Rangelands 14:35–8.

Monzingo, D.L. Jr. 1985. The prevalence of *Giardia* in a beaver colony and the resulting environmental contamination. Ph.D. dissertation. Colo. State Univ., Fort Collins, Colo. Cited in C.P. Hibler and C.M. Hancock. 1990. Waterborne giardiasis. *In* Drinking water microbiology: progress and recent developments. G.A. McFeters (editor). Springer-Verlag, New York, N.Y., pp. 271–93.

Ong, C.S.L., D.L. Eisler, A. Alikhani, V.W.K. Fung, J. Tomblin, W.R. Bowie, and J.L. Isaac-Renton. 2002. Novel *Cryptosporidium* genotypes in sporadic cryptosporidiosis cases: first report of human infections with a cervine genotype. Emerging Infectious Diseases 8:263–8.

Ong, C.S.L., D.L. Eisler, S.H. Goh, J. Tomblin, F.M. Awad-El-Kariem, C.B. Beard, L. Xiao, I. Sulaiman, A. Lal, M. Fyfe, A. King, W.R. Bowie, and J.L. Isaac-Renton. 1999. Molecular epidemiology of cryptosporidiosis outbreaks and transmission in British Columbia, Canada. Amer. J. Tropical Medicine and Hygiene 61(1):63–9.

Ong, C., W. Moorehead, A. Ross, and J. Isaac-Renton. 1996. Studies of *Giardia* spp. and *Cryptosporidium* spp. in two adjacent watersheds. Appl. Environ. Microbiol. 62:2798–2805.

Ongerth, J.E., G.D. Hunter, and F.B. DeWalle. 1995. Watershed and *Giardia* cyst presence. Water Res. 29:1295–9.

Packer, P.E. 1953. Effects of trampling disturbance on watershed condition, runoff and erosion. J. For. 51:28–31.

Palmateer, G. 1997. New method to recover *Cryptosporidium* and *Giardia* from drinking water. Environ. Sci. Eng. May 1997.

Radostits, O.M., K.E. Leslie, and J. Feltrow. 1994. Herd health: food animal production medicine. 2nd ed. W.B. Saunders, Philadelphia, Pa., pp. 312–4. Cited in Centre for Coastal Health. 1996. Summary report of the key aspects of *Cryptosporidium parvum* with special reference to cattle. Dept. of Health Care and Epidemiology, Univ. B.C., Vancouver, B.C.

Rendtorff, R.C. 1954. The experimental transmission of human intestinal protozoan parasites: II. *Giardia lamblia* cysts given in capsules. Am. J. Hyg. 59:209–20. Cited in J.L. Isaac-Renton. 1987. Giardiasis: a review. B.C. Med. J. 29:341–4.

Rose, J. 1988. Occurrence and significance of *Cryptosporidium* in water. J. Amer. Water Works Assoc. 80(2):53–8.

Rose, J.B. 1990. Occurrence and control of *Cryptosporidium* in drinking water. *In* Drinking water microbiology: progress and recent developments. G.A. McFeters (editor). Springer-Verlag, New York, N.Y., pp. 294–321.

Rose, J.B., H. Darbin, and C.P. Gerba. 1989. Correlations of the protozoa, *Cryptosporidium* and *Giardia*, with water quality variables in a watershed. Water Sci. Tech. 20:271–6.

Rose, J.B., C.N. Haas, and S. Regil. 1991. Risk assessment and control of waterborne giardiasis. Amer. J. Public Health 81:709–13.

Rose, J.B., J.T. Lisle, and M. LeChevallier. 1997. Waterborne cryptosporidiosis: incidences, outbreaks, and treatment strategies. *In* *Cryptosporidium* and cryptosporidiosis. R. Fayer (editor). CRC Press, Boca Raton, Fla., pp. 93–109.

San Francisco Public Utilities Commission. 1996. *Cryptosporidium* white paper. City and County of San Francisco, Calif.

Sherer, B.M., J.R. Miner, J.A. Moore, and J.C. Buckhouse. 1988. Resuspending organisms from a rangeland stream bottom. Amer. Soc. Agric. Eng., St. Joseph, Mich. Paper No. 88-2082.

_____. 1992. Indicator bacterial survival in stream sediments. J. Environ. Qual. 21:591–5.

Smith, H.W. and J.B. Rose. 1990. Waterborne cryptosporidiosis. Parisitology 6:8–12.

Sneva, F.A. 1969. Behavior of yearling cattle on eastern Oregon range. USDA and Oreg. State Univ. Agric. Exp. Stn., Corvallis, Oreg. Oregon State Univ. Tech. Pap. No. 2624. Cited in R.E. Larsen. 1989. Water quality impacts of free ranging cattle in semi-arid environments. M.Sc. thesis. Oreg. State Univ., Corvallis, Oreg.

Soave, H.V. and D. Armstrong. 1986. *Cryptosporidium* and cryptosporidiosis. Rev. Infect. Diseases 6:1012–23. Cited in M.R. Barer and A.E. Wright. 1990. A review: *Cryptosporidium* and water. Letters in Appl. Microbiol. 11:271–7.

Springer, E.P. and G.F. Gifford. 1980. Unconfined grazing and bacterial water pollution—a review. *In* Symp. on watershed management 1980. Vol. 1. Amer. Soc. Civil Engineers, Irrigation and Drainage Div., pp. 578–87.

Sterling, C.R. 1990. Waterborne cryptosporidiosis. *In* Cryptosporidiosis of man and animals. J.P. Dubey, C.A. Speer, and R. Fayer (editors). CRC Press, Boca Raton, Fla., pp. 51–8. Cited in Centre for Coastal Health. 1996. Summary report of the key aspects of *Cryptosporidium parvum* with special reference to cattle. Dept. of Health Care and Epidemiology, Univ. B.C., Vancouver, B.C.

Sweeten, J.M. and D.L. Redell. 1978. Non-point sources: state-of-the-art overview. Trans. Amer. Soc. Agric. Eng. 21:1048–87. Cited in R. Thelin and G.F. Gifford. 1983. Fecal coliform release patterns from fecal material of cattle. J. Environ. Qual. 12:57–63.

Thelin, R. and G.F. Gifford. 1983. Fecal coliform release patterns from fecal material of cattle. J. Environ. Qual. 12:57–63.

Tiedemann, A.R., D.A. Higgins, T.M. Quigley, H.R. Sanderson, and D.B. Marx. 1987. Responses of fecal coliform in streamwater to four grazing strategies. J. Range Manage. 40:322–7.

Tiedemann, A.R., D.A. Higgins, T.M. Quigley, H.R. Sanderson, D.B. Marx, and C.C. Bohn. 1988. Bacterial water quality responses to four grazing strategies—comparisons with Oregon standards. J. Environ. Qual. 17: 492–8.

Van Donsel, D.J. and E. Geldreich. 1971. Relationships of salmonellae to fecal coliforms in bottom sediments. Water Res. 5:1074–87. Cited in R. Thelin and G.F. Gifford. 1983. Fecal coliform release patterns from fecal material of cattle. J. Environ. Qual. 12:57–63.

Wallis, P.M. 1994. Abiotic transmission—is water really significant? *In Giardia*: from molecules to disease. R.C.A. Thompson, J.A. Reynoldson, and A.J. Lymbery (editors). University Press, Cambridge, England, pp. 99–125.

Wallis, P.M., S.L. Erlandsen, J.L. Isaac-Renton, M.E. Olson, W.J. Robertson, and H. van Keulen. 1996. Prevalence of *Giardia* cysts and *Cryptosporidium* oocysts and characterization of *Giardia* spp. isolated from drinking water in Canada. Appl. Environ. Microbiol. 62:2789–97.

Wegrzyn, J.G. 1988. *Giardia* in Colorado muskrats: prevalence, host-specificity and cross-species transmission. Ph.D. dissertation, Colo. State Univ., Fort Collins, Colo. Cited in C.P. Hibler and C.M. Hancock. 1990. Waterborne giardiasis. *In* Drinking water microbiology: progress and recent developments. G.A. McFeters (editor). Springer-Verlag, New York, N.Y., pp. 271–93.

Williams, O.R. 1981. *Giardia* and the waterborne transmission of giardiasis, a general review. USDA For. Serv., Fort Collins, Colo. WSDG-TP 0003. Cited in C.C. Bohn and J.C. Buckhouse. 1985. Coliforms as an indicator of water quality in wildland streams. J. Water and Soil Conservation. 40:95–7.

Willoughby, B. 1993. Health. *In* Forestry-drinking water quality interactions: final workshop report. T. Berry, T. Webb, and C. Perrin (editors). ESSA Environmental and Social Systems Analysts Ltd., Vancouver, B.C., pp. 7–8.

Zurbrigg, M. 1992. The factors involved in determining bacterial water quality and the application to grazing management in the B.C. interior. B.C. Min. For., Kamloops Region, Kamloops, B.C. Unpublished.

Fifteen individuals were invited to participate in the interview process. Fourteen individuals participated; one person declined. An attempt was made to ensure balanced representation of viewpoints; however, not all stakeholders are represented, nor are the group sizes equal. The interviews were conducted in 1997.

Some participants asked to remain anonymous so we chose not to disclose the identity of any of the respondents when reporting their answers to the interview questions. Numbers of responses to individual questions do not always total 14 because some respondents chose not to answer certain questions.

In your opinion, is there sufficient evidence that livestock use within community watersheds can be linked to the incidence of waterborne diseases? What evidence do you view as supporting this opinion?

Five respondents said "yes," four said "no," three said "there could be," and two said they were unsure. The respondents who answered "yes" cited the Cranbrook outbreak of cryptosporidiosis as evidence of a link between cattle and waterborne disease outbreaks. The other respondents gave a variety of answers as to why they remained unconvinced of such a link. Some acknowledged that while it seems reasonable that cattle could contribute to disease outbreaks, other potential sources of contamination have often been present in areas of outbreaks. Respondents also noted that *C. parvum* and *G. lamblia* have been found in water supplies throughout North America, even in watersheds where there is no livestock grazing. Additionally, respondents stressed that even though *C. parvum* and *G. lamblia* occur in cattle, there currently are no scientific data to show that cattle are a source of the disease in humans. For instance, both *Cryptosporidium muris* and *C. parvum* infect cattle, but only *C. parvum* is known to cause disease in humans. Simply finding *Cryptosporidium* in waters where cattle are present does not necessarily indicate a threat to humans. One interviewee said they were not aware of any studies that showed that pathogens (*C. parvum* and *G. lamblia*) found in water sources were even viable and capable of causing disease in humans.

***What are the organisms of most concern* (Giardia, Cryptosporidium, Campylobacter, *other*)?**

Cryptosporidium parvum was most often cited (13 responses), followed by *G. lamblia* (11 responses), and *Campylobacter* (seven responses). Respondents were concerned about these organisms mainly because of recent disease outbreaks in the province. It was also noted that because these organisms are found in humans, wild animals, domestic animals, and surface waters, the potential for disease transmission can be substantial. Additionally, interviewees were concerned about *C. parvum* because there currently is no effective treatment for the disease cryptosporidiosis.

Many respondents acknowledged that they were most concerned about *C. parvum* and *G. lamblia* because these were the organisms with which they were most familiar. They noted that there were probably other organisms that should be of concern, but they did not know which ones those might

be. One respondent said that all zoonotic diseases were of concern. Another interviewee also expressed concern about coliforms, *Yersinia enterocolitica*, *Entamoeba histolytica*, and *Aeromonas hydrophila* because they have been found in human, animal, and water sources, and there is serological evidence of infection by some of these organisms in humans.

What management actions would you suggest to reduce or eliminate the risk of waterborne disease linked to livestock use of community watersheds?

Most responses dealt with cattle access to watersheds (eight responses), timing of cattle use of watersheds (five responses), control of surface runoff throughout the watershed (four responses), or use of buffer zones in riparian areas (three responses).

With respect to cattle access, three respondents suggested that cattle should be banned from community watersheds, while another five recommended only restricting cattle access from riparian areas in watersheds. One person noted that cattle should especially be prevented from grazing near water intakes. Another respondent, however, believed that threats to riparian areas can be reduced through proper distribution of cattle throughout the watershed. It was suggested that this can be achieved through salting and range riding, and by planning pasture use so as to avoid sensitive areas, and to avoid cattle concentrations in areas of high runoff.

Respondents also suggested that timing of cattle use of community watersheds could be adjusted to reduce the risk of waterborne diseases linked to cattle. They noted that it was particularly important to delay timing of cattle turn-out in watersheds until calves are past the peak of susceptibility to diseases such as cryptosporidiosis. Three interviewees stated that cattle should not be in community watersheds when the ground is frozen or the soil is saturated, as this increases the risk that contaminated feces will be transported into water sources by spring runoff.

Four respondents suggested that the risk of waterborne diseases linked to livestock use of community watersheds could be reduced through the use of livestock management practices that did not lead to increased surface runoff. They did not state, however, what these practices might be. Presumably, they were referring to maintaining vegetation cover and preventing soil compaction in upland and riparian areas.

Respondents also recommended using riparian buffer zones or alternative watering methods (e.g., water troughs) to reduce cattle access to drinking water sources. Two respondents suggested that if the link between cattle and waterborne diseases was verified, then drinking water sources should be fenced. Other interviewees noted that fencing water sources will not eliminate pathogens from drinking water supplies, since aquatic mammals and other wild animals may also be sources of waterborne diseases.

One respondent stressed that, before restrictions on cattle use of watersheds are considered, animals should be tested to determine disease prevalence in herds. If levels of infection are cause for concern, then emphasis should be placed first on reducing or eliminating the disease in the herd, and second, on restricting access to water sources in watersheds.

Other suggestions for reducing the potential risk to drinking water from

cattle included: preventing disease transmission to cattle herds; avoiding cattle concentrations (e.g., calving, feeding, overwintering sites) in areas that have surface runoff to watercourses; and improving water treatment methods. One respondent also suggested that a total review of watershed usage was needed in the province. They recommended that personnel be hired on a full-time basis to monitor cattle use of community watersheds so that good baseline data could be gathered. This would lead to a better understanding of livestock use of those areas, and would provide information that could be used in developing risk assessment plans for watersheds. In addition to these suggestions, one interviewee stated that guidelines for issuing grazing permits in community watersheds need to be formulated.

Two respondents stated that the best way to ensure that cattle do not contribute to waterborne disease outbreaks is to properly implement provincial community watershed guidelines for livestock use of riparian areas. A document by the B.C. Ministry of Forests (1997) clarifies the application of the community watershed guidelines to range management activities.

Besides livestock, what are other potential sources of waterborne disease present in community watersheds (in your geographic location, or to your knowledge)?

Interviewees most often named humans (nine responses) and wildlife (eight responses). Recreational activities in watersheds, improper or poorly maintained sanitation facilities in logging and mining camps, and leaks or spills from residential septic tanks or sewage systems were identified as the main sources of human fecal contamination of community water sources. It was also noted that domestic animals (e.g., dogs and horses) accompanying people on recreational activities in watersheds can also be sources of water contamination.

Beavers and muskrats were most often identified as wildlife sources of waterborne diseases. Beavers often build dams and lodges near water intakes. Increased pathogen levels near the intake may result from direct fecal deposition in the water, or from runoff flows that create turbulence in beaver ponds and cause bottom sediments to be disturbed. One respondent also noted that the potential for wild ungulates and rodents to contribute to waterborne diseases is not known in British Columbia. Another respondent stressed that removing livestock from community watersheds will not eliminate the risk of waterborne diseases, since more wild animals may move into the watershed after cattle are removed.

What are the potential exacerbating factors to the spread of waterborne diseases by livestock (in your geographic location, or to your knowledge)?

The most common answers were: disease transmission between animals (four responses); erosion, sedimentation, and runoff patterns (four responses); watershed characteristics (three responses); and livestock management activities (three responses). One respondent questioned whether imported beef cattle were tested for *C. parvum* upon entry into the province.

According to respondents, mining activities, forestry activities (e.g.,

road-building, clearcutting), and livestock management activities are the primary sources of erosion, sedimentation, and elevated runoff in community watersheds. High runoff levels following snowmelt or storm events are often correlated with increased concentrations of waterborne pathogens. Runoff events cannot only transport pathogen-laden feces and sediments on land into watercourses, they can also create turbulence within the stream, causing pathogens trapped in bank ar ' bottom sediments to be released back into the water column. High runoff levels can also create erosion and increased sedimentation of water sources. Reducing the health risk of pathogens in turbid waters can be difficult. In such cases, chlorine treatment of water is often ineffectual. Disease organisms tend to cling to sediment particles in the water, and so are best removed through filtration. Communities that rely solely on chlorine treatment of drinking water may be more susceptible to outbreaks of waterborne diseases. Two respondents believed that improper or inadequate water treatment could be a contributing factor in the spread of waterborne diseases.

Respondents also noted that watershed characteristics such as soil types and drainage patterns can play a role in the spread of waterborne diseases by affecting the concentrations of pathogens transported into the watercourse. For example, disease organisms are more likely to be trapped in fine-sediment than in coarse-sediment soils. In the watercourse itself, water depth and waterbody connectivity can also affect concentrations of disease organisms. For example, in shallow waters, pathogens can be deactivated by ultra-violet radiation, while in watercourses with a series of deep ponds, disease organisms tend to settle out in bottom sediments. Long watercourses may also have lower pathogen levels than short watercourses because there is greater opportunity for organisms to settle out in sediments before they reach the water intake.

In terms of livestock management, interviewees stated that poor management practices in and around riparian areas could contribute to the spread of waterborne diseases. Erosion of stream banks due to trampling, elevated runoff and stream sedimentation levels due to the removal of vegetation by grazing in upland and riparian areas, deposition of fecal material in and near streams, grazing intensity, and location of calving sites near water sources were all named as potential contributing factors in the spread of waterborne diseases. One respondent also noted that vegetation removal through wildlife grazing could contribute to elevated runoff and sedimentation levels in watersheds. One interviewee stated that peaks in disease infectivity also affect the spread of waterborne diseases.

In your opinion, is there any concern regarding other forest practices (besides livestock grazing) within community watersheds related to the incidence of waterborne diseases?

Although some respondents stated that they were unaware of any scientific studies that directly linked forestry activities with waterborne diseases, most interviewees noted that practices that cause erosion and sedimentation, changes in watershed hydrology, and changes in human, wildlife, and livestock use of community watersheds could all potentially contribute to

outbreaks of waterborne diseases.

Road-building and vegetation removal through logging were cited as the main factors contributing to increased access by humans, wildlife, and livestock to riparian areas in community watersheds. In terms of recreational use of watersheds, increased access not only affects the number, but also the type, of people who use the area. One respondent believed that watersheds with easy access routes tended to draw recreationists who were not aware of, or were less conscientious about, the impacts of their activities on local water quality.

Changes in vegetation cover through seeding of clearcuts or through forest succession following logging can also cause changes in wildlife and livestock distribution in watersheds. If these animals are vectors for disease transmission, then changes in their use patterns could affect levels of pathogens in water sources, and rates of disease transmission among animals. One respondent also noted that sanitary facilities in forestry camps can be sources of water contamination, especially if they are not properly operated or maintained. Individual forestry workers can also contribute to water contamination if they do not follow proper waste disposal procedures when working in the woods.

There was a difference of opinion among some interviewees about the extent to which forestry activities could contribute to waterborne disease outbreaks. One respondent believed that impacts from clearcutting had a greater effect on water contamination than livestock management activities. Other respondents thought that improvements had been made in reducing the impacts of forestry activities on water quality, and that improvements would continue to be made, provided that provincial guidelines were followed.

What water sampling procedures would aid in the detection of waterborne disease spread by livestock, or provide evidence of causation between waterborne disease and livestock?

Five respondents did not provide answers to this question due to lack of experience with this subject. Two other respondents noted that it is difficult to make any recommendations because the science of water sampling is still in its infancy. Four respondents recommended that better means of sampling untreated water are needed.[4] Six respondents also stressed that on-going water sampling is needed to effectively monitor water quality. Some respondents thought that sampling at regular time intervals should be done, but others stressed that it would be more effective if sampling efforts were concentrated around periods of high runoff (e.g., due to snowmelt or storm events), since outbreaks of waterborne diseases often correlated with peaks in runoff. Respondents believed that this type of sampling program would

4 The Membrane Filter Dissolution Technique reportedly provides average recovery rates of approximately 60% for *C. parvum* and *G. lamblia*, and up to five samples can be processed in 1 day (Aldom and Chagla 1995; Palmateer 1997). An additional procedure can be used to test the viability of *C. parvum* oocysts. The complete process of recovery, identification, and viability testing takes 48 hours. Conversely, the traditional Environmental Protection Agency water sampling method has a recovery rate of only 2–10% for *C. parvum* and *G. lamblia*, and takes 2½ days to analyze one sample. Additionally, one of the drawbacks of traditional water sampling methods is that there is no way to determine if pathogens recovered are viable (Palmateer 1997).

require a more intensive sampling effort for short periods, but would produce better information about prevalence of waterborne diseases than would less frequent regular sampling over longer periods.

Other recommendations for sampling included: use of genetic markers to track disease sources and transmission routes; increased sampling of cattle, other livestock animals, and wildlife to track disease prevalence; and increased routine sampling of treated water to determine effectiveness of water treatment methods, especially around periods of disease outbreaks. One respondent also stressed that whatever sampling methods and programs are implemented, proper training of technicians must be made a priority to ensure that accurate and reliable results are obtained.

What supplementary information would aid in determining the cause of outbreaks of waterborne disease?

Interviewees believed that the collection of the following information would aid in determining the cause of waterborne disease outbreaks:

- weather conditions and streamflow levels prior to, and during, disease outbreaks;
- history of land use practices in community watersheds prior to disease outbreaks (e.g., types of forestry activities, types and levels of recreational use, livestock numbers and distribution, and timing of access);
- changes in land use practices in watersheds prior to disease outbreaks; and
- wildlife species, numbers, and distribution in watersheds prior to disease outbreaks.

In your judgement, what is the best approach to resolve the controversy regarding causes of waterborne disease in community watersheds (e.g., research, workshops, extension, other)?

Most interviewees (nine) believed that the best way to resolve the controversy about causes of waterborne disease outbreaks in the province was through education. A few of the interviewees held the opinion that British Columbia has the highest incidence of waterborne diseases in Canada, yet there is a public perception that British Columbia's drinking waters are pristine. As a result, there has been little public pressure or political will to fund appropriate water treatment facilities in many communities throughout the province. It was suggested that all water users need to be educated about the various sources of disease contamination of drinking water, and of the importance of protecting drinking water supplies.

Most respondents thought that projects such as our literature review were an appropriate first step in the education process. They also suggested that the best way to get the message out to the public about protecting drinking water from waterborne diseases was through open houses, workshops, extension courses, brochures, newspaper articles, public displays in shopping malls, field trips, television coverage of the issue, and signs in watersheds. Demonstration areas showing appropriate forest and livestock management practices around riparian areas were also seen as an effective means of show-

ing that these activities, if properly managed, do not necessarily have negative impacts on drinking water quality.

One respondent believed that individuals should be educated about taking responsibility for treating their own water supplies. This person noted that the cost of water treatment programs is often considered prohibitive by many levels of government. Other respondents (five) stated that a public lobby for clean drinking water is needed to educate politicians about the necessity of committing funding for proper water treatment programs throughout the province. One interviewee suggested that if water use fees were raised to reflect the importance of the resource, the extra funds generated could be directed towards implementing effective water treatment programs.

Other respondents thought that protecting drinking water sources through proper management of forestry and livestock practices and human access was either at least as important, or more important, than implementing water treatment programs. Other respondents emphasized that it is impossible to have pristine watersheds (e.g., wild animals can be potential vectors of disease transmission), and thus unreasonable to expect zero risk of contamination at the source of drinking water supplies.

Two respondents noted that water needs and watershed use differ among communities, so local solutions to local problems are needed. They thought the best way of addressing individual community situations was by developing local water management plans, or by using watershed risk assessment methods.

A few respondents stated that the best way to resolve the controversy surrounding waterborne diseases in British Columbia was to identify knowledge gaps and research needs regarding the issue.

One respondent was pessimistic that anything could be done to resolve the controversy around livestock use of watersheds and potential links with waterborne disease outbreaks. This person noted that this is often an emotional issue, and public perceptions are so well entrenched that no amount of good scientific data will change those perceptions. Another respondent stressed that those perceptions, rather than science, are currently driving the debate over proper management of British Columbia's drinking water sources.

What research needs on the topic of waterborne disease and forest practices do you consider important, if any?

Respondents recommended that the following topics be investigated, or that more local information on these subjects be gathered:

- effectiveness of water treatment systems in controlling levels of waterborne pathogens;
- identification of more reliable indicators of water quality (fecal coliform concentrations are considered to be poor indicators of *C. parvum* and *G. lamblia*. *Clostridium* may be a better indicator for such pathogens [Berry et al. 1993]);
- development of standardized methodology for detecting waterborne pathogens;

- concentrations of waterborne *C. parvum* necessary to create outbreaks in the human population;
- life cycles of pathogens in different host species;
- ecology of waterborne pathogens, including their movement, viability, and survivability from the host source through the entire water system to the tap;
- sources of waterborne pathogens, and their significance in outbreaks of waterborne diseases (one way to accomplish this may be through the use of genetic markers);
- transmission of waterborne diseases between different hosts;
- impacts of forestry activities on incidences of waterborne disease outbreaks;
- prevalence of diseases in different wildlife species throughout British Columbia;
- prevalence of diseases in cattle herds throughout British Columbia;
- age at which calves develop some resistance to *C. parvum*;
- prevalence of disease-resistant genes in livestock that have access to community watersheds;
- impacts of cattle in riparian areas in local watersheds (e.g., rates of manure deposition in and near streams, and the impact on water quality);
- methods to reduce impacts of cattle in riparian areas (e.g., use of alternative watering systems); and
- associations between cattle management practices and outbreaks of waterborne diseases. These should be good, quantifiable studies in which cattle presence and movements in watersheds are tracked, and details on grazing season, intensity, and duration are correlated with pathogen levels in water supplies, and with outbreaks of waterborne diseases.[5]

Respondents stressed the need for local, long-term studies in British Columbia. They noted that most information on the subjects of livestock impacts in riparian areas, and incidences of waterborne diseases, has come from studies in U.S. watersheds. Results of these studies may not be applicable to conditions in British Columbia. Two respondents suggested that the best way to study cattle impacts in riparian areas is to establish paired-watershed research areas. Watershed characteristics and past resource use should be similar between paired watersheds. At least one watershed would be used as a control, while other watersheds could be used for experiments on grazing management practices. Respondents stated that this would not only provide local answers to local questions, but could also be used to teach about the effects of particular livestock management activities in watersheds.[6] One

5 In addition to these recommendations, studies on the length of time fecal material acts as a source of contamination need to be determined to properly assess the impact cattle have on water quality (Springer and Gifford 1980). The Environmental Protection Agency (1979) further notes that information on grazing season, duration, and intensity, and on local site conditions, is needed to properly assess grazing effects on water infiltration and runoff on rangelands. Quantitative relationships between grazing intensity, utilization, trampling, and infiltration rates remain undetermined for most plant communities (Environmental Protection Agency 1979).

6 Similar research areas could be used to study the effects of forestry activities on waterborne diseases (Berry et al. 1993).

respondent stated that we need to take a wider perspective with our research projects than just studying "the pathogen of the day." This person noted that at any time there could be an outbreak of a more serious pathogen than has been previously encountered. This tends to result in research money being withdrawn from on-going studies and being re-directed to the organism of latest concern. This respondent recommended that we should focus our research efforts more on protecting our water resources than on trying to control individual pathogens.

List of Interviewees

B.C. Ministry of Agriculture, Fisheries and Food[7]

Dr. Mervyn Wetzstein	Health Management Veterinarian

B.C. Ministry of Environment, Lands and Parks

Dr. Rick Nordin	Water Quality Branch
Larry Pommen	Water Quality Branch
Ron Townson	Southern Interior Sub-Regional Office
Dr. Pat Warrington	Water Quality Branch
Ted White	Water Quality Branch

B.C. Ministry of Forests

Doug Fraser	Forest Practices Branch – Senior Range Practices Agrologist
Grant Griffin	Cranbrook Forest District – Range Officer
Bruce Johnson	Prince George Forest Region – Regional Range Officer
Alex McLean	Penticton Forest District – Range Officer

B.C. Ministry of Health

Vicki Carmichael	Environmental Health Assessment and Safety Branch

Other Health Organizations

Ken Christian	South Central Health Unit #6, Kamloops, B.C.
Dr. Bill Moorehead	South Okanagan Health Unit #5, Kelowna, B.C.

University of British Columbia Faculty of Medicine / B.C. Centre for Disease Control

Dr. Corinne Ong	Pathology and Laboratory Medicine

7 Listed affiliations are those provided at the time of the interview in autumn 1997.

APPENDIX 2 Scientific names of wildlife species mentioned in text

Common name	Scientific name
beaver	*Castor canadensis*
black bear	*Ursus americanus*
black rat	*Rattus rattus rattus*
cottontail rabbit	*Sylvilgagus nuttalli*
coyote	*Canis latrans*
Dall's sheep	*Ovis dalli*
deer	*Odocoileus* spp.
deer mouse	*Peromyscus maniculatus*
domestic cat	*Felis domesticus*
domestic cattle	*Bos* spp.
domestic dog	*Canis familiaris*
domestic pig	*Sus scrofa*
domestic sheep	*Ovis aries*
elk	*Cervus elaphus*
grizzly bear	*Ursus arctos*
horse	*Equus caballus*
house mouse	*Mus musculus domesticus*
lynx	*Lynx canadensis canadensis*
marten	*Martes americanus*
mink	*Mustela vison*
mule deer	*Odocoileus hemionus*
muskrat	*Ondatra zibethicus*
Norway rat	*Rattus rattus norvegicus*
raccoon	*Procyon lotor*
red fox	*Vulpes fulva*
striped skunk	*Mephitis mephitis*
white-tailed deer	*Odocoileus virginianus*
wolf	*Canis lupus*

B.C. Ministry of Agriculture, Fisheries and Food[8]

Dr. Mervyn Wetzstein, 1998	Health Management Veterinarian

B.C. Ministry of Forests

Doug Fraser, 1998	Forest Practices Branch
Grant Griffin, 1998	Cranbrook Forest District

B.C. Ministry of Health

Barry Boettger, 1998	Water Quality Consultant

Other Health Organizations

Dr. Bill Moorehead, 1998	South Okanagan Health Unit #5, Kelowna, B.C.
Dr. Gillian Arsenault, 1999	Medical Director / Medical Health Officer Fraser Valley Health Region Previously: East Kootenay Community Health Services Society
Don Corrigal, 1999	East Kootenay Community Health Services Society

B.C. Ministry of Municipal Affairs and Housing

Eric Bonham, 1998	Municipal Engineering Services

8 Listed affiliations are those provided at the time of the personal communication (1998–1999).